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Table of Contents

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ORIGINAL ARTICLES— PAGE.

The Surgical Sequelæ of Acute Cerebral Trauma, by GILBERT PHILLIPS, M.Sc., M.B., B.S. 41
The Sequelæ of Head Injuries, by C. G. McDONALD, M.B., Ch.M. 45
Acute Upper Abdominal Pain, by J. COLVIN STOREY, M.B., Ch.M., F.R.C.S. 52

REPORTS OF CASES—

Local and General Anaphylaxis, by ROBERT S. STEEL, M.B., Ch.M., M.R.C.P., and DAVID ADCOCK, M.B., Ch.M. 59

REVIEWS—

Diseases of the Nervous System 59
Varicose Veins and Hæmorrhoids 60
Living Things 60

LEADING ARTICLES—

Occupational Therapy and its Modern Development 61

CURRENT COMMENT—

Chordoma 62
The South African Journal of Medical Sciences .. 63

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.

Morbid Anatomy 64
Morphology 65

BRITISH MEDICAL ASSOCIATION NEWS—

Scientific 66
Nominations and Elections 71

OBITUARY—

Leila Katherine Keatinge 71

CORRESPONDENCE—

Problems of Junior Resident Medical Officers .. 71

BOOKS RECEIVED 71

MEDICAL APPOINTMENTS 72

MEDICAL APPOINTMENTS VACANT, ETC. 72

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 72

EDITORIAL NOTICES 72

THE SURGICAL SEQUELÆ OF ACUTE CEREBRAL TRAUMA.[1]

By GILBERT PHILLIPS, M.Sc., M.B., B.S.,

Honorary Neurological Surgeon, Lewisham Hospital; Lecturer in Surgery, University of Sydney.

LESIONS which arise as the sequelæ of acute cerebral trauma may be conveniently considered from the point of view of their situation. Although all of them are intracranial, one group is extracerebral and the other intracerebral. It is impossible here to discuss fully the ætiology, the clinical picture and the treatment of these two groups, so that the subject will be considered in general terms, while at the same time reference will be made from time to time to aspects of each lesion which are of particular interest.

The extracerebral group of lesions succeeding cerebral trauma is due to injury to blood vessels. The consideration of middle meningeal hæmorrhage which is a rare complication of cerebral trauma does not concern the present discussion. The common vascular sequela of cerebral trauma is subdural hæmorrhage, which in over 90% of cases is of venous origin. Subdural hæmorrhage is about three times as common as extradural hæmorrhage and may take place either between the dura and the arachnoid or into the subarachnoid space.

The classical situation in which such hæmorrhages occur is from the cortical veins which pass medially and slightly forwards to join the superior longitudinal sinus at the supero-medial border of each cerebral hemisphere. (Figure I.) Each of these veins is anchored at one end to the cerebrum by tributary veins passing upwards from the sulci, and at the other end by the junction with the superior longitudinal sinus. The vein is, as it were, "strung out" between these points, and relative movements taking place between them may succeed in stretching or tearing these veins. Such movement does occur when the cerebral hemispheres are thrown violently

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on October 31, 1935.

forward or backward as a result of frontal or occipital trauma. As each vein passes medially it lies at first in the subarachnoid space; it then pierces the arachnoid and after a short course between the arachnoid and the dura it enters the sinus. As the vein may be torn either before or after piercing the arachnoid the hæmorrhage may be either subdural and extraarachnoid or subdural and subarachnoid. Hæmorrhage in the latter situation is the more frequent, and it may safely be said that some blood is always extravasated into the cerebro-spinal fluid in moderate and severe cerebral trauma. Such extravasation is always a concomitant of laceration or severe contusion.

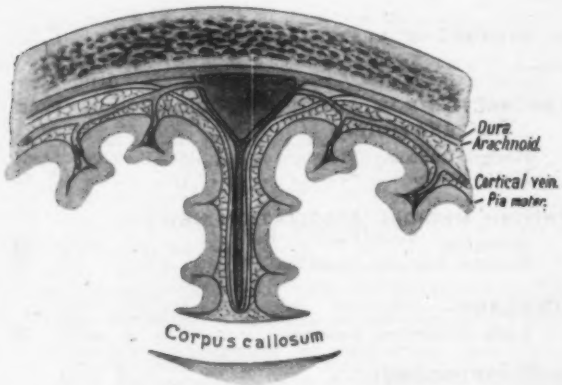

FIGURE I.

Diagram showing the relations of the cortical veins to the arachnoid and dura. The vein is anchored at one end to the cerebrum by its tributaries, and at the other to the superior longitudinal sinus. It may be torn before it pierces the arachnoid or while it is coursing medially between the arachnoid and the dura.

The clinical picture presented by subdural hæmorrhage may take three forms, and appears to depend on two factors, namely, the situation of the hæmorrhage and the amount of blood extravasated. In one form there may be present a massive frontal clot, either subarachnoid or extraarachnoid in situation. (Figure II.) Such hæmorrhage usually results from severe injury, the patient is usually unconscious *ab initio* and remains unconscious until the clot is removed. If the hæmorrhage takes place on the inferior surface of the frontal lobes, and particularly if the lesion is bilateral and accompanied by cerebral laceration, the patient usually, after a period of unconsciousness, becomes delirious or even maniacal owing to interruption of inferior frontal inhibition to the autonomic centres in the hypothalamus. Such cases are often fatal.

In the second form the hæmorrhage is subarachnoid and of small or moderate volume. The injury is moderate, the patient may be unconscious for a few hours only, and is usually treated as though he were suffering from cerebral contusion. Recovery from the original trauma is generally uneventful and the patient is discharged from hospital either perfectly well or with some residua of the cerebral contusion (motor defects). Some months later he returns with a history of intermittent attacks of headache and dizziness of sudden onset, often succeeding physical effort or slight trauma and accompanied perhaps by increase in the disability (weakness *et cetera*) if such exists as a result of the original injury. If no disability exists these cases particularly may simulate the picture of post-traumatic neurosis, since complaint may be made of loss of interest in the previous occupation, irritability, failing memory *et cetera*. It is usual, however, when the original lesion was a small subarachnoid hæmorrhage for the patient to describe remissions between the attacks which have recently become more serious. Unconsciousness for a short period may have occurred in a recent attack. On examination, papillœdema of low degree may sometimes be detected.

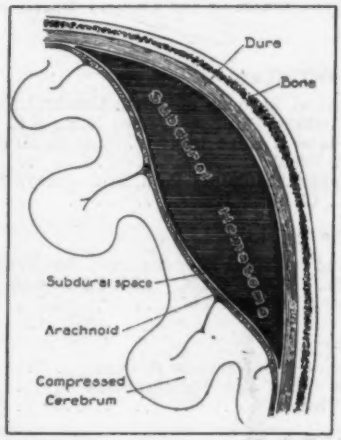

FIGURE II.

Diagram showing the relation of a subdural hæmatoma to the dura and the pia-arachnoid and the manner in which such a collection of blood can compress the cerebrum. If such compression is permitted to persist, it is inevitably followed by neural destruction, neurogliosis and cerebral scarring. (After Zollinger and Gross.)

The sequence of events in these cases has been as follows: The subarachnoid lake on the supero-lateral convexities of the hemispheres is shallow and is divided into a maze of little compartments by the trabeculæ which join the outer surface of the pia to the inner surface of the arachnoid. When blood is extravasated here from a cortical vein it spreads through the maze, displacing the cerebro-spinal fluid before it. When hæmorrhage ceases, as it characteristically does spontaneously in these cases, the blood clots and a blood plaque or one form of subdural false membrane is formed. (Figure III.) Delicate capillaries and young fibroblasts grow into this plaque during the succeeding weeks, and then suddenly as the result of some slight trauma one or more of the small delicate vessels in the plaque are ruptured and a further hæmorrhage occurs. The patient has a sudden headache, feels giddy, and if the plaque overlies the motor cortex, weakness of a contralateral limb may develop or in some cases epileptiform seizures of the focal type may ensue.

This series of events tends to be repeated at intervals until the false membrane is removed.

The third form of subdural hæmorrhage takes place between the arachnoid and the dura. It is less common than the preceding forms, and is usually the result of mild injury. It is often bilateral, and in a typical case described recently bilateral lesions of this type were produced in a man whose occupation was that of a roof inspector. He had not experienced any recent head injury, but in the course of his occupation he habitually bumped up trapdoors with his head to gain access to the roofs which it was his duty to inspect.

FIGURE III.

Photomicrograph showing the early formation of a membrane about a collection of blood between the arachnoid and the dura (Dew and Phillips, unpublished). Two cubic centimetres of blood were injected between the dura and arachnoid of a macacus monkey. Seventy-two hours later the membranes enclosing the clot were excised and sections made. This section shows red and white blood corpuscles above, and several laminæ of spindle-shaped fibroblasts growing in the deepest layers of the hæmatoma. The dura and arachnoid are not seen here.

Blood extravasated between the dura and the arachnoid shows little tendency to clot, and it may extend on either side from the frontal to the occipital pole. The clinical picture in these cases is characteristic. After some mild head injury the patient returns to his occupation to find that he is not up to his original standard. He will suffer from headaches and giddiness and may complain of a sense of unreality. A few months after the original injury the headaches commence to become severe, nausea and vomiting follow, and there may be mild disturbances of motor power and of the tendon reflexes, particularly in the lower limbs. On examination bilateral papillœdema can often be detected. The symptoms and signs are progressive and it may be difficult to decide whether the lesion is a neoplasm or a subdural hæmorrhage. Only recently has a reasonable explanation been provided for the progressive nature of the symptoms in these cases. Zollinger and Grosse have shown that a membrane is formed about the extravasated blood and that this membrane has semi-permeable properties (Figures IV and V). At first the contained blood is only slightly hypertonic to the cerebro-spinal fluid and the blood cyst increases slowly in size by the passage of cerebro-spinal fluid through the membrane. The extravasated corpuscles begin to disintegrate and liberate into the sac the molecules which they contain. The osmotic tension inside the hæmatoma rises rapidly and cerebro-spinal fluid is drawn rapidly through the membrane. The hæmatoma displays a progressive increase in size, and in this way behaves as a neoplasm.

The operative treatment of this form of hæmatoma is simple. A small burr hole is made over the frontal cortex 2·5 centimetres (one inch) from the mid-line. Through this opening a small nick is made in the dura. Chocolate coloured old blood which has not coagulated can then be seen and a second burr hole and second nick in the dura are made about four centimetres posterior to the first. By irrigation and suction through these two openings the old blood is washed out. It is not necessary to remove the membrane. It should be remembered that the condition is often bilateral. As the cerebro-spinal fluid pressure is often low in these cases, an osteoplastic flap should not be turned down, as subsequent capillary oozing may reform the hæmatoma.

The intracerebral sequelæ of cerebral trauma are produced by the destruction of nerve cells and nerve fibres. It has recently been shown that when nerve cells, even in such widely separated areas as the *nucleus dentatus* of the cerebellum and the frontal

FIGURE IV.

Photograph (after Zollinger and Gross) showing a subdural hæmatoma, enclosed in its membrane, removed *en bloc* from its situation between the arachnoid and the dura.

cortex, are injured, they become absorbed and are then replaced by neuroglial cells. If nerve cells or fibres are contused, there is inevitably set up the following train of events: Contusion, softening and absorption, neuroglial proliferation, contraction of the resulting cerebral scar. The extent and degree of this process depend entirely on the intensity of the original contusion. During the stage of softening blood vessels may become involved and may rupture. In this way a rare sequela of cerebral trauma is produced. This condition, known as *Spätapoplexie*, was described many years ago. The onset is sudden and occurs characteristically from ten to fourteen days after the head injury. It may

occur while the patient is at rest or during physical effort. This type of hæmorrhage usually takes place deep in the white matter and frequently ruptures into the lateral ventricle. For this reason many of these cases are fatal. The most common site of cerebral contusion is in the frontal poles, so that the clinical picture in *Spätapoplexie* is usually the abrupt onset of hemiplegia, with or without aphasia, in a patient convalescing from some moderate cerebral trauma received from ten to fourteen days previously.

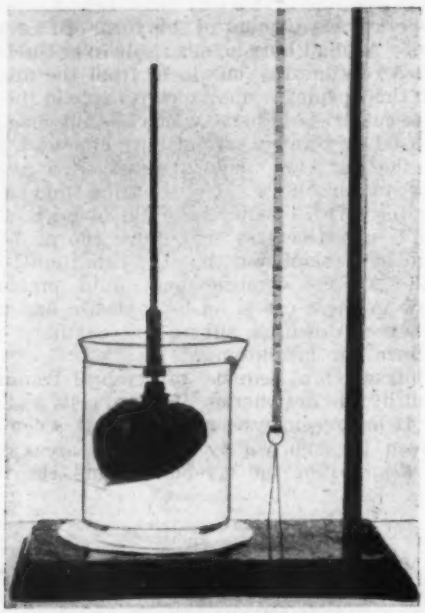

FIGURE V.

Photograph (after Zollinger and Gross) showing the manner in which the membrane of a subdural hæmatoma behaves as a semi-permeable membrane. Inside the membrane is placed oxalated whole blood; in the beaker is a normal saline solution. A rise of pressure in the manometer tube of 15·5 centimetres took place in twenty-four hours.

Neurogliosis is not infrequently the cause of epilepsy succeeding cerebral trauma. The trauma may have been so mild that it is often not even considered as a possible cause of the convulsions. The convulsions may not appear for many years (an interval of ten years is reported later), and since the patient is usually quite well in the intervening period, the head injury may have been forgotten when the convulsions suddenly begin. Neurogliosis may follow mild cerebral trauma, but post-traumatic epilepsy is usually the sequel of a head injury of moderate or severe degree. Post-traumatic convulsions are ten times more common when the dura is penetrated by depressed bone. The cerebral scar which is produced by neurogliosis is usually situated in the pre-frontal region. As it grows older it contracts and exerts traction on neighbouring nerve cells, notably those in the motor cortex. The length of the period between the original head injury and the resulting convulsions depends on the severity of the original trauma and the proximity of the traumatized area to the motor cortex. Pathologically, a characteristic lesion is produced. There is an orderly arrangement of the proliferating neuroglia which is laid down at right angles to the surface of the cortex. (Figure VI.) As the scar commences to contract, the surface of the cerebral hemisphere is drawn inwards and at operation a large pool of cerebro-spinal fluid is found overlying the defect. At the same time the ventricle wall is drawn outwards by the contracting scar (Figures VII and VIII). The resultant traction diverticulum of the ventricle is of fundamental diagnostic importance, since in a suspected case of post-traumatic epilepsy the diagnosis may often be made absolute by taking skiagrams after encephalography by the lumbar route (Figures X, XI and XII). Convulsions succeeding cerebral trauma may be Jacksonian in type or may be generalized from the beginning. If the former, the pattern of the fits is very constant and is repeated exactly during subsequent seizures. If the latter, there may or may not be present some localizing sign such as deviation of the head and eyes to the opposite side. (Figure XIII.)

FIGURE VI. Schematic drawing of three levels in any meningo-cerebral cicatrix of long standing. The uppermost panel shows blood vessels and bundles of collagen penetrating the brain beneath the meninges. The middle panel, from an area farther below the surface, shows intimate relationship of collagen strands and fine-fibred neuroglia, all parallel and pointing in the direction of the cicatricial pull. The lowermost panel, from an area deeper in the brain, shows the structure of astrocytes and blood vessels which make up the vaso-astral framework and transmit the cicatricial pull. (After Penfield and Foerster.)

Surgical treatment of these cases involves wide and deep excision of the scar down to and into the lateral ventricle. It has been shown by Foerster and Penfield that if this is performed radically the scar does not reform.

In conclusion may I repeat the words of Lennox and Cobb used in an extensive thesis on epilepsy published from the Department of Neuropathology of the Harvard Medical School:

> Many writers have segregated patients who present no significant abnormality on physical examination, and whose seizures, therefore, are presumably due to an inherent tendency to react with convulsions, into a group labelled "Essential" or "Idiopathic". Because it seems probable that all patients having seizures from whatever apparent cause possess this

susceptibility to seizures in some degree, and because we have no means of determining whether it forms 10% or 90% of the total influences making for seizures, the clinical use of such a term as "essential" only obscures the issue, making discovery of the underlying cause or causes more difficult. Some disturbance of the brain exists in all cases in which seizures occur.

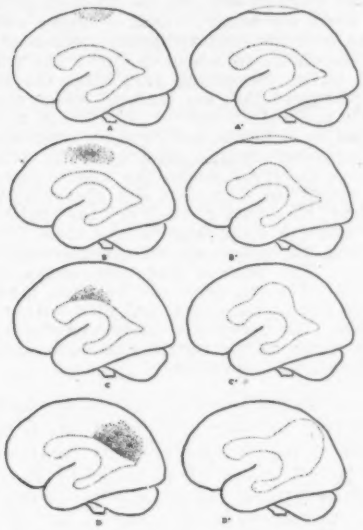

FIGURE VII.

Diagram illustrating the method by which the cerebro-spinal spaces compensate for any loss of brain tissue. A: defect in or near the cortex is compensated by dilatation of the superimposed subarachnoid spaces; A′, B: defect midway between the ventricle and the surface of the brain is compensated by dilatation of the subarachnoid space over the defect and ventricular dilatation under the defect; B′, C: defect deeply seated in the brain is compensated by dilatation of the ventricle; C′, D: atrophy of the whole thickness of the brain is compensated by a great diverticulum of the ventricle; in some cases this may assume tremendous proportions. (After Dandy.)

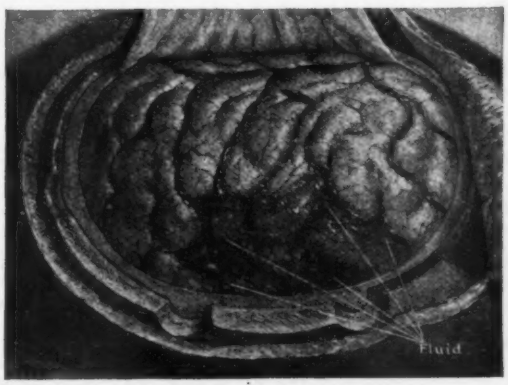

FIGURE VIII.

Operative finding in typical case of epilepsy resulting from cerebral atrophy. The localized bed of fluid in the subarachnoid space is evidence of space compensation for the loss of cerebral tissue. (After Dandy.)

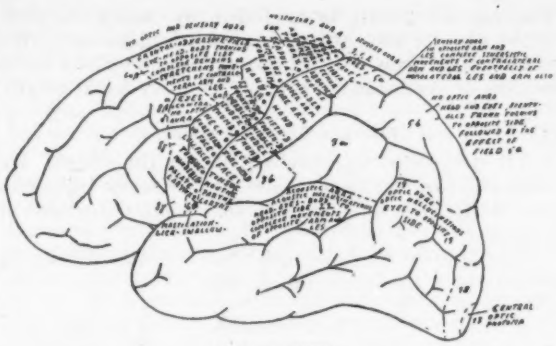

FIGURE XIII.

Epileptogenic cortical areas in the human brain. The outlines indicate roughly the initial movements and auræ which follow direct stimulation of the cortex by an electrical current sufficient to produce an epileptic seizure. (After Penfield and Foerster.)

(1) G. Jefferson: "The Treatment of Acute Head Injuries", *The British Medical Journal*, November 4, 1933, page 807.
(2) H. W. B. Cairns: "The Treatment of Head Injuries", *The Lancet*, March 17, 1928, page 566.
(3) Ernest Sachs: "Head Injuries", *International Journal of Medicine and Surgery*, December, 1933, page 567.
(4) R. Zollinger and R. E. Gross: "Traumatic Subdural Hematoma", *The Journal of the American Medical Association*, Volume CIII, July 28, 1934, page 245.
(5) Bollinger: *"Über traumatische Spät-Apoplexie: ein Beitrag zur Lehre von der Hirneschütterung", Internationale Beiträge für Wissenchafliche Medizinische Festschrift*, Volume II, 1891, pages 457 to 470.
(6) Wilder Penfield and Otfried Foerster: "The Structural Basis of Traumatic Epilepsy and the Results of Radical Operation", *Brain*, Volume LIII, 1930, page 99.
(7) Walter Dandy: "Studies in Experimental Epilepsy", *Bulletin of the Johns Hopkins Hospital*, Volume XXXVI, January, 1923, page 245.
(8) Walter Dandy: "The Space Compensating Function of the Cerebro-Spinal Fluid; Its Connection with Cerebral Lesions in Epilepsy", *Bulletin of the Johns Hopkins Hospital*, August, 1923, page 245.
(9) Wilfred Trotter: "Chronic Subdural Hæmorrhage", *The British Journal of Surgery*, Volume II, October, 1914, page 271.
(10) G. E. Phillips: "Radiography in the Diagnosis of Intracranial Tumours", *The Australian and New Zealand Journal of Surgery*, July, 1934, page 30.

THE SEQUELÆ OF HEAD INJURIES.[1]

By C. G. McDONALD, M.B., Ch.M. (Sydney),
Lecturer in Clinical Medicine, University of Sydney; Honorary Physician, Royal Prince Alfred Hospital, Sydney.

ALTHOUGH the surgeon is better equipped to discuss the immediate results of injury to the skull and brain, the physician has perhaps a more intensive experience of the later effects of this damage. Into his consulting room come those obstinate patients who have left the shelter of hospital, apparently healed of their wounds, but not yet cured of all their symptoms. Of many of these patients the surgeon has long since grown tired and many are destined to try sorely the more enduring patience of the physician.

It is customary to divide the sequelæ of head injuries into two main groups: those dependent on organic damage to the brain and meninges, and those regarded as neurotic or "functional" in origin.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on October 31, 1934.

The second group is included by many English writers under the caption "traumatic neurosis". We might well form a third group characterized by a medley of symptoms of organic and psychical origin.

Primary Cranial Lesions.

All symptoms, whether organic or functional, are dependent on the occurrence of a primary injury to the skull and its contents in varying degrees of gravity.

The injury may be any one of the following lesions or a combination of them:

1. *Concussion.*

Concussion is a sudden and generalized compression of the brain, lasting only a fraction of a second, but sufficient to produce paralytic symptoms of apparent and sometimes of real gravity. That concussion itself may kill without any visible laceration, contusion or hæmorrhage of the brain, has long since been established. Marked as it is by all the phenomena of shock (pallor, vasomotor paralysis), by complete loss of consciousness, complete relaxation of the whole body, paralysis of the sphincters and light, shallow breathing, it is one of the best examples we have of the results of a profound anæmia of the whole brain, or at least of that portion which lies above the tentorium.

2. *Extradural, Subdural, Subarachnoid or Intracerebral Hæmorrhage of Gross Type.*

Extradural Hæmorrhage.—Extradural hæmorrhage occurs most frequently in connexion with fractures of the base or vertex involving the middle cranial fossa, the vessel commonly torn being the middle meningeal artery. Recovering from the deep unconsciousness of concussion, the patient improves for a time and then sinks again into coma as a result of cerebral compression by blood which bursts from the ruptured artery and strips the dura from the bone. Contralateral paralysis of the face and arm, convulsions and even aphasia, if the lesion is left-sided, occur before the unconsciousness becomes deep. Fluid withdrawn by lumbar puncture is clear.

Subdural Hæmorrhage.—Subdural hæmorrhage, commonly held to be the most frequent type of traumatic bleeding, is of interest to the physician because he is familiar with its manifestations under the name of *pachymeningitis hæmorrhagica interna.* Rupture of a small vessel under the dura may occur after trivial blows and the slowly developing hæmorrhage, limited by the formation of a fibrinous barrier, may produce symptoms so slow in their development and so undramatic in their manifestations, that the condition is often mistaken for a traumatic neurosis. This so-called "chronic type" of subdural hæmorrhage is responsible for a persistent complaint of increasing headache and sometimes for mental changes which simulate hysteria.

CASE I.—A man was knocked down by a motor car as he was crossing a road. He was dazed slightly, but acquired no wound on his head. It is not even known whether his head struck against car or road. He was taken to hospital, where the casualty surgeon was concerned chiefly with an injury to one leg and asked him to return the following day for X ray examination of this limb. Next morning the patient complained of headache. The pain persisted, but was evidently not severe, as after four days he returned to work. There was a gradual change in his behaviour during the succeeding fortnight. At work and when at home in the evening, he kept calling out: "My head, my head!" waved everyone aside, became morose and irritable, and ultimately demanded to see his grown-up daughter, to whom, he told his wife, he had just given birth. Seventeen days after the accident his wife was convinced that her husband was a sick man and sent for his medical attendant. The latter, a sound practitioner, finding no abnormal signs on examination of the nervous and other systems, and evidently believing that the man was hysterical, asked the wife to leave the room and then closed the door. But she, observing at the keyhole, beheld the doctor shaking the patient by the shoulders and exhorting him to be a man. The medical attendant did not call again till urgently summoned four days later. The patient was now semi-conscious and obviously ill. He was sent into hospital, gradually sank into deeper and deeper coma, and died within two weeks of admission. The pathologist found at autopsy a subdural hæmatoma which he regarded as traumatic and of at least three or four weeks' standing.

Gross subdural hæmorrhage is marked by little or no interval between the symptoms of concussion and those of severe hemispheral compression. In this, as in all cases of tension on the meninges, headache of violent character is a characteristic symptom before coma ensues, and in the short reactionary interval mental excitement and maniacal delirium may dominate the clinical picture.

Subarachnoid Hæmorrhage.—Subarachnoid hæmorrhage differs in no essential from the subdural type. The presence of much blood in the cerebro-spinal fluid in a suspected case of meningeal hæmorrhage aids the diagnosis.

Intracerebral Hæmorrhage of Gross Type.—Intracerebral hæmorrhage of gross type can be diagnosed only in cases of cerebral compression following on concussion and cerebral irritation if the hæmorrhage involves those areas of the brain capable of clinical localization. The early development of hemiplegia or of hemianopsia before the onset of coma is always suggestive of an intracerebral lesion.

CASE II.—The motor car in which Doris P., aged twenty-seven, was a passenger on October 12, 1934, crashed into a telegraph pole. She regained consciousness in the Western Suburbs Hospital three weeks later. On coming to, she was told and she noticed that she was paralysed in the left arm and the left leg. She had no feeling in the left side of her face, and sensation was dulled in the left arm and left leg. Eight months after her accident the left upper limb is still useless. She cannot use it to dress and she cannot hold a cup in her left hand. She walks with a pronounced limp, swinging the left leg outwards and forwards, and inclining her body slightly to the left with each advance in walking. She moves, however, unassisted and without the aid of crutch or stick.

On examination (eight months after the injury) slight but definite paresis of the left side of the face is evident. The grossest change is present in the left hand. Quite fair power and control exist in the left arm and forearm, but the hand is virtually useless. The hand is held fully extended at the wrist and the fingers are fully extended. With extreme difficulty she lifts a light blotting pad off

my table, but immediately it falls from her hand. The paralysis is of the spastic type. The deep reflexes of the left upper limb are grossly exaggerated. The left lower limb is paretic. There is obvious weakness of the iliopsoas and quadriceps muscles and of the extensors of the foot. The paresis is spastic, although the left knee jerk is not greatly increased. The left leg is blue and cold, contrasting with the right. Sensation to pin prick is dulled in the left arm, left leg and left side of the trunk. It is normal in the left side of the face. There is marked impairment of the sense of passive position and passive movement in the toes of the left foot. There is no Rombergism.

This young woman had no mental impairment and there was a refreshing absence of any of the psychical symptoms which are so common after head injuries. She had no headache, no dizziness, no sleeplessness, no fear of traffic. She complained of the symptoms of hemiplegia and of those alone. Her blood pressure readings were 135 and 85 millimetres of mercury.

3. *Severe Cerebral Trauma without Gross Hæmorrhage.*

In severe cerebral trauma without gross hæmorrhage the patient fails to recover consciousness after the primary concussion and passes into a state of cerebral irritation or, worse still, into deep coma, but manifests no localizing signs of compression. There may be an associated fracture of the base, but sometimes, as in fatal cases recorded by Cassasa in America, and recently by Berner in Scandinavia, death may result without any evidence of fracture. Cassasa described punctiform hæmorrhages in the vicinity of the basal ganglia, while Berner found similar lesions in the medulla and pons.

The assumption in all these cases is that scattered hæmorrhages and small areas of contusion occur above or below the tentorium. If the brain stem is seriously affected by these small lesions, paralytic bulbar symptoms make their appearance and the patient dies rapidly. If the patient recovers, the absence during his stay in hospital of any localizing signs may beguile the surgeon into the belief that the patient is neurotic or is prolonging his period of incapacity for his own ends.

4. *Traumatic Encephalopathy ("Punch Drunk").*

Closely allied to the preceding type of injury is the condition found in professional boxers whose ability to take punishment is greater than their skill to avoid it. "Punch drunk" fighters are men who have sustained repeated concussion with apparent recovery from each attack, but in whom clinical evidence of disseminated cerebral lesions becomes manifest as the years pass by. It would seem that there is some virtue in a fractured skull, for a boxer who sustains a fracture may take the hint and abandon what Gene Tunney described as a dangerous occupation. "Punch drunk" was well known to *habitués* of the ring for years before its grudging recognition by neurologists. Martland drew attention to it in 1928 and emphasized its importance. He suggested that a violent blow on the head caused a commotion in the cerebro-spinal fluid, which was driven with great force into the prolongations of the subarachnoid space which penetrate the cerebral hemispheres along with the cerebral vessels, and that the momentum of the fluid ruptured the minute vessels at points where trabeculæ pass from the vessel wall across the subarachnoid space to the pia-arachnoid. In other words, Martland's suggestion is that the pathology of "punch drunk" is identical with the "ring" (or peri-vascular) hæmorrhages within the brain which Cassasa described in fatal cases of concussion. However, whatever the pathology, no doubt can exist of the clinical entity of this condition. Punch drunk fighters commonly suffer from slurring speech with considerable difficulty in pronouncing consonants, from mild ataxia, from tremor of head or limbs, and from a stiffness and clumsiness of one or both feet. Many of them have unilateral nerve deafness. The majority are simple, almost childish, fellows, and some find their way ultimately into mental asylums. The clinical manifestations are progressive and the suggestion has been made that a slow gliosis of the brain develops in the areas where the vascular damage is greatest. In Cassasa's fatal cases the "ring hæmorrhages" were deep seated in the cerebral hemispheres, none being apparent in the cortex and the majority occurring in and around the basal ganglia. According to Martland, the majority of punch drunk boxers resemble patients suffering from *paralysis agitans*, but the greater number of those whom I have examined had signs which simulated disseminated sclerosis.

The significance from the neurological point of view of "punch drunk" (now glorified with the long and ugly name of "traumatic encephalopathy") is that we have here definite organic stigmata of repeated but apparently mild and transitory attacks of concussion without fracture of the skull.

5. *Cortical or Subcortical Hæmorrhage with Focal Signs.*

After recovery from the primary concussion the patient may manifest cerebral irritation, but instead of passing into the stage of cerebral compression he may quickly or slowly present signs of a cortical or subcortical lesion. The site of the lesion is made evident by the occurrence of monoplegia, with or without Jacksonian convulsions, or of aphasia, agraphia or alexia, or of incomplete hemianæsthesia or of hemianopsia. The hæmorrhage occurs under the point of impact. If the hæmorrhage is cortical, the arachnoid mesh prevents its spread. These lesions are not uncommon after gunshot wounds and after direct injury to the skull with an instrument of small striking surface.

CASE III.—On March 31, 1917, a young Australian officer sustained a gunshot wound of his right upper arm while directing the operation of a machine gun in a trench in France. Suffering severe pain, he remained at his post. Two hours later a bullet struck him just above and just in front of the left ear. In his own words, he did not think much of this second wound. He did not lose consciousness. He rose from the kneeling position and sat down. He noticed that he could now move his right arm without pain and that he was completely anæsthetic down the right side (face, arm and leg). Within three minutes of the second injury, believing that the Germans might be coming over to attack, he called out a warning to a soldier

with a machine gun at the end of the trench. Noises of many kinds came from his mouth, but not a single word. He knew exactly what he wanted to say, but could not say it. He realized that he was making only ridiculous sounds (motor aphasia). "I then had a 'brain wave', took a pencil out of my tunic and determined to write 'Fight the machine gun!' on the wall of the trench. I wrote a lot of K's and L's and F's, all mixed consonants and no vowels. They were good letters, but they made nonsense, and I knew it. The soldiers thought I had gone silly" (agraphia). At dusk he was taken out of the trench. The stretcher-bearers thought he was almost dead, and he groaned to let them know that he was much alive. At the casualty clearing station he had time to consider his condition. He had severe pain in the left side of the head. He tried by gestures to stop the barber from shaving his head, as it was painful and tender. The barber completed his task and said to the theatre attendants: "Put him to one side; he will be dead in the morning." This remark annoyed him, as he knew he was not going to die. But he could not speak. He was still anæsthetic down the right side. He had no motor paralysis. That evening he was operated on. A small piece of steel was removed from the temporal bone, which was not completely penetrated. X ray examination revealed no fracture. He was in bed for three weeks. He was still unable to speak, but still able to understand every word spoken to him. He received letters from home, but could not read them. He could see the letters, knew that they made words, knew that the words had meaning, but did not know what the meaning was (alexia). Two weeks after his injury he wrote a letter to his mother with considerable difficulty, and a month later he wrote one which had some, but not many, faults in spelling. Thus he wrote "aslo" instead of "also". In convalescence he experienced considerable difficulty in eating, for he could not use his right hand for cutting, because, he believes, he could not tell what he was feeling with his right hand. The knife would slip out of his hand, his forefinger slipping off the back of the knife. "I lost my fine sense of touch." Gradually he recovered. At first he recovered his ability to read, and in three months he could talk as well as formerly. Meanwhile the right-sided anæsthesia had disappeared.

Instructive as this story is, the aftermath is of equal interest.

In August, 1917, on the transport returning him to Sydney, he was shaving one morning when he noticed sudden loss of feeling down the right half of his body (face, trunk, arm and leg) and loss of motor speech as well. He recovered quickly. On arrival in Australia he suffered from attacks which were the sensory equivalent of Jacksonian fits. In these attacks the right foot became numb and soon the "loss of feeling" worked half way up the right leg. Then a tingling sensation appeared in the right leg and spread upwards into the right arm and thence into the right side of the face and into the right eye. He compares the feeling in the right eye to the irritation caused by tobacco ash. Soon the right arm became painful and at the same time he felt a burning sensation in the right side of the face. This burning became very intense. No difficulty in speech or writing occurred. When these attacks subsided his right hand was anæsthetic for some time.

About a year after his return to Australia he noticed that attacks similar to those just described were followed after an interval by what he describes as "epilepsy in the right hand". The whole of the right hand, including the fingers (the forearm was not involved) "jumped about as if subject to electric shocks". At the same time he felt intense tingling in the hand and fingers. Then almost immediately he lost his speech. He could neither speak, read nor write, and for the first time he had difficulty in understanding the speech of others (sensory aphasia). He could hear them, could hear the words, knew that they were speaking, but did not know what the words meant. Further, in these attacks he was unable to use a pen or pencil or to handle a knife of fork, or even to put on his coat or open a door, although he knew exactly what all these objects were for (apraxia). If these attacks came on when he was in town, he would decide to go quietly home, avoiding recognition by friends. He had no difficulty in recognizing persons and could walk wherever he willed. These attacks still occur, but come rarely. In the interim he has the appearance of a normal man.

A consideration of this case leads to no other conclusion than that the gunshot wound caused either a small subcortical hæmorrhage or an area of subcortical contusion. The retention of consciousness is evidence of the absence of compression, and the complete recovery between attacks is proof of the small size of the lesion. Further, the occurrence of repeated attacks of sudden onset and equally sudden disappearance, characterized by Jacksonian motor phenomena and their sensory equivalents and by apraxia and motor and sensory aphasia, are proof of the profound local disturbance of the circulation which may occur on and off for many years after a cerebral injury. What is the nature of this disturbance and why is it ephemeral and yet recurrent? In the damaged area of the brain a blood vessel may lie, its neuromuscular irritability heightened, it may be, by a traumatic lesion of its wall. It needs a comparatively small stimulus to evoke local spasm of such a vessel. Whether the stimulus is chemical or physical or even psychical, we do not know, but it has been stated, and it is conceivable, that abnormal factors external to the brain may provoke an attack of *grand mal* in an epileptic person. It is at least permissible in the present poverty of our knowledge to adopt as an hypothesis the conception of local spasm in a vessel with temporary ischæmia of the part supplied. Perhaps the more slowly developing and more widespread signs are due to the formation around the ischæmic region of a widening field of œdema which obstructs the blood flow in the surrounding veins.

6. *Gross Local Injury of the Skull and Brain by Direct Violence.*

In many cases of gunshot wounds gross local damage to the skull and brain may occur. There is an open wound, and a compound depressed fracture of the skull occurs. This type of injury is of great importance to the surgeon on account of the risk of sepsis, but it also has an indirect interest for the physician in that serious signs of cerebral damage are usually absent, owing to the relief of tension within the cranium by the open wound in the skull. It is unnecessary to labour here the principle established as far back as 1896 by Leonard Hill, that all cerebral symptoms are fundamentally due, neither to spicules of bone nor to clots of blood nor to tumour masses, but to the associated anæmia of the brain produced by displacement of venous and then of arterial blood by the entry of these foreign bodies into the "incompressible box" of the cranium.

Sequelæ of Undoubted Organic Origin.

No physician has any difficulty in attributing to organic cerebral damage certain well-defined symptoms or signs. These include monoplegia and hemiplegia of organic type; epileptic convulsions, whether generalized or Jacksonian; apraxia,

Norm
the

ILLUSTRATIONS TO THE ARTICLE BY DR. GILBERT PHILLIPS.

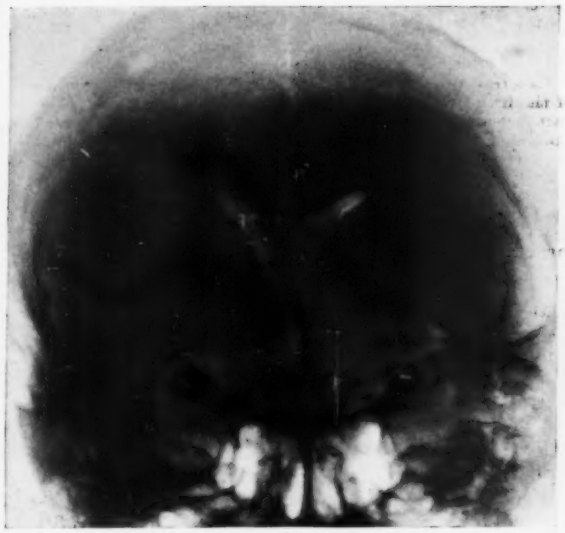

FIGURE IX.

Normal encephalogram. Antero-posterior skiagram showing the typical "butterfly" appearance of the anterior horns of the lateral ventricles. (Phillips.)

FIGURE X.

Antero-posterior skiagram after encephalography, showing large traction diverticulum in the anterior horn of the left lateral ventricle. This patient, a man aged twenty-six, had been struck with a stone on the left frontal region at the age of six. Generalized convulsions with head-turning movements to the contralateral side commenced after an interval of ten years at the age of sixteen.

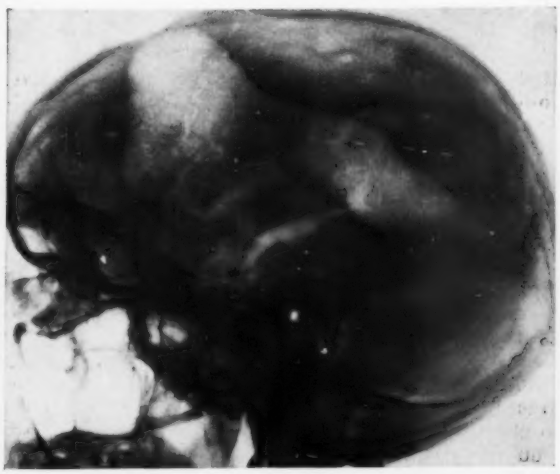

FIGURE XI.

Lateral skiagram of the same case (Figure X).

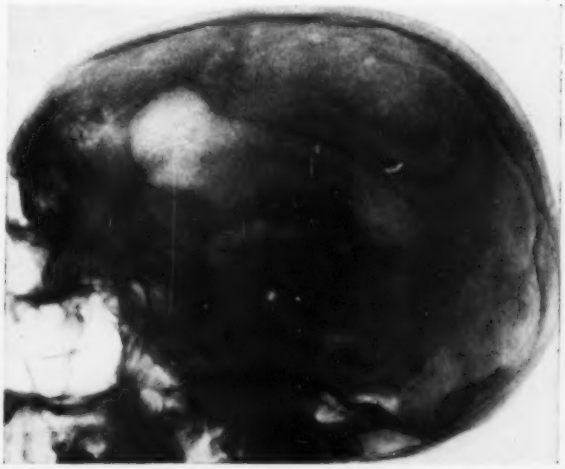

FIGURE XII.

Lateral skiagram after encephalography in a patient who developed generalized convulsions some years after being involved in an automobile accident. Note the traction diverticulum in the anterior horn of the left lateral ventricle.

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aphasia, alexia, agraphia and dysarthria; insanity or obviously serious change in mental disposition; signs of disseminated cerebral lesions; nerve deafness; and occasionally isolated cranial nerve symptoms, such as diplopia, anosmia and loss of the sense of taste. Of these we need speak no further, but it is necessary to emphasize that symptoms of organic origin may be present when careful physical examination of the nervous system fails to reveal any abnormal sign. The case described above, of the soldier who sustained a gunshot wound of the skull in 1917, is an illustration of this important fact, for in the intervals between attacks no abnormality of his nervous system can be discovered. Further, there are symptoms entirely subjective which may have an organic basis, even when they simulate closely the complaints of neurotic persons. Such symptoms are: headache, dizziness or giddiness, loss of the power of mental concentration, forgetfulness and mental deterioration of all grades up to frank insanity. If we are to believe many recent writers, these symptoms are invariably the direct result of material injury to the brain. That in certain cases they are truly organic sequelæ cannot be denied. Whether we should regard them as invariably organic is, however, best dealt with under the next heading.

The Subjective Symptoms Following Head Injury.

When a patient, complaining of such purely subjective symptoms as pain in the head, dizziness, fatigue after effort and lack of the power of concentration, presents himself before the physician, the latter is confronted with one of the most difficult problems in medicine. He must decide whether residual damage is present in the brain or whether the symptoms have an entirely psychical origin. There is little doubt that prior to 1924 the "post-concussion syndrome" was held by the majority of physicians to be dependent on psychical factors alone. The term "traumatic neurosis" meant that the symptoms emanated from no pathological change in the brain. In that year, however, Trotter published his well known paper, "On Certain Minor Injuries to the Brain", in which he described gross changes in the cerebral substance years after the occurrence of the injury. "It is surprising", he states, "how long after the injury the signs of contusion will remain evident in the injured area. I have myself exposed by operation an area of brain that has been bruised by the glancing contact of a bullet with the skull no less than four years earlier. The brain showed a bruise that appeared as fresh as if it had been inflicted within a few weeks. This inordinate and unique delay in the resolution of contusions is fully adequate to account for the striking persistence of traumatic headaches." To Trotter's view, not only headache, but dizziness, fatigue after effort, and all other subjective symptoms are due to alterations in intracranial tension by persistent cerebral contusions. His lead has been followed by many others. Recently Strauss and Savitsky, after investigation of many patients by means of encephalograms and an imposing array of clinical tests, which included a comparison of the intraocular tension and of the diastolic pressure in the retinal arteries of the two eyes, examination of the visual fields, the Muck adrenaline test, and a test for "resorption" of cerebro-spinal fluid by injecting two cubic centimetres of a 10% solution of sodium iodide into the spinal theca, and noting the time of the appearance of iodine in the urine, came to the conclusion that "the subjective post-traumatic syndrome . . . is organic and is dependent on a disturbance in intracranial equilibrium due directly to the blow on the head".

Now these opinions, if true, are disturbing, for they mean that those of us who reassure many of our patients and encourage them to return to the active duties and responsibilities of life, even after a minor injury to the head, are not acting in their best interests and are in fact helping to delay the already protracted resolution of persistent cerebral contusions. But the same criteria apply to clinical neurology as to general medicine, and the evaluation of a patient's symptoms remains a clinical problem when pathological or experimental evidence is unconvincing. No one denies that violence applied to the skull may result years later in the detection at autopsy of meningeal adhesions, arachnoid cysts and areas of cerebral softening and atrophy, but how many surgeons would be bold enough to search for the contusion which is responsible for a patient's "traumatic neurosis"? It is an inviting question to ask how frequently surgeons have located in the brain, even one year after injury, "a bruise that appeared as fresh as if it had been inflicted within a few weeks". A physician cannot deny the occurrence of persistent cerebral contusions, and a man of Trotter's exalted standing as a surgeon must always be heard with respect. But a physician is at liberty to draw inferences which the clinical history renders inevitable.

CASE IV.—Two months ago a young time-keeper on the Sydney wharves struck the top of his head against an iron link as he was walking along the deck of a steamer. The scalp was not injured. He felt no ill-effects and went on working. Half an hour later he slipped down a stairway leading from the upper to the lower floor of a wharf at Darling Harbour. He remembers slipping on the staircase, but does not remember reaching the bottom. He was momentarily stunned. He sat up and then stood up, feeling dazed. Soon he felt nauseated, but did not vomit. A small hæmatoma appeared on the scalp, in the upper temporo-parietal region, but there was no open wound. After resting for fifteen minutes he resumed work. All day he felt dizzy and inclined to vomit, and his head was aching. Next morning, on his way to work, he fell over. He does not know what happened. He rose at once, felt much better, except for dizziness, and continued on his way. During the morning his headache returned, and at noon he consulted a doctor, who diagnosed "slight cerebral concussion" and sent him for one week into a private hospital. After a further week's rest at home he returned to work, but after one day had to cease on account of frontal headaches and weariness from insufficient sleep. After three weeks' further rest he worked for three weeks, but yesterday ceased duty once more, as he could no longer bear the headaches, the sleeplessness and the weariness. He averages only three hours' sleep each night.

Inquiry elicits the fact that he was not enjoying good health when he sustained "concussion". Two years ago he was afflicted with domestic worries. His father was a drunkard and an intolerable quarreller. The son was required to support the whole family with the meagre assistance of a young sister who had just commenced work. The responsibility was too great for a young man of weak character. Sleep became broken and difficult, and soon he was unable to sleep at all for three or four nights in succession. He became nervous, irritable and weary, he lacked concentration and his work was inefficient. He persevered for twelve months and then consulted a medical man, who prescribed potassium bromide. His employers sent him on a sea trip to Western Australia, lasting three weeks, after which he camped for a week on the Nepean River. After nine weeks' absence he returned to work, improved in health, but still troubled with insomnia. Such was his condition when his head was injured two months ago.

He was a thin, small young man. The only abnormalities were found on examination of the nervous system. There were rather slow choreiform twitchings of various parts of the body, especially the shoulders. The twitchings were more evident when he was excited, for example, when his blood pressure readings were being taken. Exaggeration of the knee jerks was the only other abnormal sign.

In this case the patient's symptoms are due to psychical factors which operated prior to his accident. The blow on the head and the fall down the stairway half an hour later were unfortunate in that they suggested to his mind the possibility that he had sustained a grave injury to his brain. In other words, his condition was one of anxiety neurosis.

CASE V.—A rubber worker, aged twenty-two years, was walking through a factory on September 6, 1935, when he suddenly fell. He does not know whether he slipped or tripped over an object. All he remembers is a hard hit on the right side of the forehead as he fell. The forehead struck one of the tire moulds stacked against the wall of the factory. He was dazed. He rose and then sat down on a box, his forehead bleeding. First aid was administered and he was then dispatched in an ambulance to a public hospital, where a superficial wound was sutured. He was then allowed to go home. Next afternoon (Saturday) he became dizzy and vomited frequently. He returned to the hospital and was admitted for three days. X ray examination revealed no evidence of fracture. On discharge from hospital his head was aching badly, he was still dizzy, and he was vomiting. A month after his accident he was still complaining of severe frontal and occipital headache, nausea and vomiting after every meal, dizziness on stooping, and a sensation of pressure in his right eye. Physical examination revealed no abnormality of the nervous system. His mentality was good.

He was encouraged to eat more food each time he vomited, and he was reassured again and again that no injury to the brain had resulted. His weight, which had depreciated 7·2 kilograms (sixteen pounds) after his accident, increased from 54·9 kilograms (eight stone ten pounds) to 60·7 kilograms (nine stone nine pounds) in two weeks, the vomiting, headaches and dizziness ceased, and he returned contentedly to work.

There can be little doubt that anxiety was the origin of this patient's symptoms. His complaints ceased with the resolution of his fears.

Deductions made from encephalograms and ventriculograms and from adrenaline and other tests must be discounted in many instances by the absence of controls. Unless an encephalogram shows incontrovertible evidence of internal hydrocephalus or ventricular displacement or of hemispherical atrophy or of meningeal adhesions, its value as evidence is suspect. Slight apparent dilatation of one ventricle and slight excess of air over one hemisphere as compared with the other are but vague evidence of intracranial damage.

Much of the criticism levelled at those who propound a psychical origin for the symptom-complex following concussion is based on objections to the view that patients who have sustained head injury either deliberately or "unconsciously" exaggerate their symptoms in order to obtain monetary compensation. But surely this conception of the cause of the traumatic syndrome is long since dead. There is no more reason why a man should simulate or unconsciously exaggerate his symptoms after an injury to the head in order to achieve personal gain than after an injury to the arm or leg. The majority of patients are honest, and few neurotic persons can be justly charged with the desire to exploit their symptoms in the market place. The cause of the neurosis is much deeper. There is a remarkable similarity between the symptoms following head injury and those following injury to the back. In both there is the same vague and distressing headache, the same complaint of dizziness, the same sleeplessness, fatigue and lack of mental concentration. There are three portions of the human body which, when injured, provoke the development of symptoms with no organic basis: the brain, the back and the left side of the chest. Man has an inherent fear of injury to his brain, his spine or his heart, and this fear is reinforced by the conversations of his friends, who invariably know of someone who after a similar injury died of cerebral hæmorrhage or was admitted to a lunatic asylum or was paralysed for life or was a cardiac cripple till he was ready for the grave. Unhappily the patient's fears are too often confirmed by his medical attendant, who fails to appreciate the mental origin of vasomotor symptoms and who assumes organic damage to exist when there is none. No physician beholding a young patient who has experienced a sharp pain in his left breast after the lifting of a heavy weight, who complains of palpitation, precordial pain, dizziness, dimness of vision on stooping, headache and insomnia, and who loses these symptoms after vigorous reassurance, can fail to appreciate the potent effect of the mind in the production of vasomotor instability and its associated symptoms. No stranger contrast in medicine exists than that between a man who faces with courage or equanimity the amputation of a limb and another who staggers, vomits and pales after a trivial tap on the head.

More surprising than the occurrence of neurotic stigmata after mild injuries to the head is their frequent absence in patients who have sustained widespread organic damage. The contention has been raised that in these cases the fracture of the skull or the actual avulsion of large areas of bone allows of adjustments in the cerebral circulation which obviate the persistence of œdema and the resultant rise in intracranial tension. The punch drunk boxer disproves this view. Here is a man who has had repeated attacks of concussion without

fracture of the skull, knocked insensible by blows which cause sufficient damage to make his speech slurring, his legs spastic and his gait staggering, and yet he is a cheerful soul, unanxious and uncomplaining. The importance of the punch drunk syndrome in the consideration of the pathology of traumatic cerebral lesions cannot be emphasized too much.

In any estimate of a patient's condition after head injury, clinical judgement must be the ultimate deciding factor. Not seldom a patient with residual organic damage is frankly neurotic and fear adds to his symptoms and to his unhappiness. But we are not justified in classing as neurotic symptoms which are capable of an organic explanation and which are accompanied by symptoms of indubitable organic origin.

CASE VI.—A labourer, aged forty-five years, was working in a brick-pit near the pit face on April 16, 1935, when a piece of shale fell from a height of approximately forty feet and struck him on the left side of the head. He has no memory of the accident or of any events for a week following. He sustained a compound comminuted and depressed fracture involving the left parietal and temporal bones and was deeply unconscious on admission to hospital. Craniotomy was performed, the left middle meningeal artery was found to be ruptured, and was ligated low down in the middle cranial fossa, the dura not being opened. The depressed fragments of bone were elevated. Rising from bed after twelve weeks, both legs, especially the left, felt weak, he was giddy and he staggered to the left when he walked. He discovered that he was stone deaf in the left ear and that noises heard in his right ear were painful (hyperacusis). Hearing in the right ear is now normal. Now, six months after the injury, his residual symptoms are deafness in the left ear, a stagger to the left when he walks, and severe diffuse headaches felt in the forehead, on the vertex and over the occiput. He does not complain of sleeplessness, and, apart from slight nervousness in traffic, he is content and confident, and feels well.

On careful physical examination no abnormality could be detected beyond the craniotomy depression, with loss of bone, slight exaggeration of the knee jerks, deafness of the left ear, and a reel to the left when he was walking. The abdominal reflexes were present. The response on plantar stimulation of both feet was flexor. There was no Rombergism, and no adiadochokinesis, past-pointing or hypotonia was present. There was no tremor.

No doubt can remain that this man sustained in addition to the fracture and the hæmorrhage from the middle meningeal artery, damage to the vestibular mechanism and to the auditory nerve on the left side. His headaches are inconstant, but it is probable that they are caused by interference with the circulation of the blood through membranes thickened by the injury. The vasomotor influence on a cranial lesion causes a variability in the intensity of the symptoms.

The significance of headache is largely determined by its nature. The narrower its localization on the skull, the more likely is it to be organic in origin. Neurotic patients who complain of pain in the head have difficulty in localizing it and lavish on its description a wealth of fantastic imagery which makes it suspect at once. Much doubt may also be cast on a pain which is uninfluenced by large doses of aspirin.

Dizziness is a symptom common to many different conditions. It is one of the most frequent complaints of patients. The convalescent rising from a prolonged stay in bed, the sufferer from essential hypertension, the student trimming his midnight lamp, the tobacco addict, the over-worked professional man, the child with measles creeping upon him—all complain of dizziness. Dizziness is no more mysterious in the victim of concussion than in the worried merchant who staggers to a nervous breakdown. At first subjective, it may ultimately develop into objective ataxia. Doubtless it is the effect of mild cerebral anæmia on a sensitive vestibular system, but the patient need not have a hæmorrhage in his semicircular canals or vestibular nuclei or along the course of the vestibular nerve. In fact, he seldom manifests any evidence of a vestibular lesion, and the otologist tests the vestibular reactions in vain. Emotional influences acting through the medium of the vasomotor system are among the commonest causes of dizziness. The truth can be expressed by saying that headache and dizziness are only rarely due to organic cerebral lesions.

Grave alteration in mental disposition after an accident is indicative of organic changes. Sleeplessness, irritability and nervousness in traffic are most commonly due to anxiety, but the development of a morose and taciturn nature in a person of previously bright disposition is an index of grave cerebral damage. It may be the herald of traumatic insanity.

Treatment.

It is happily no part of my function to discuss the surgical treatment of the sequelæ of head injuries. I need merely say that a physician should agree to surgical interference only when he is convinced that an accessible organic lesion responsible for serious symptoms exists.

The treatment of the neurotic sequelæ is the physician's special care. Much of his difficulty and many of his disappointing failures would be obviated if the attending surgeon took pains to allay his patient's gathering fears in the early days following the accident. A quiet but convincing word of comfort at this stage is worth more than hours of reassurance six months later. The magic words, "Your brain is undamaged", spoken to a neurotic patient, will resolve anxieties which are none the less terrible for being unexpressed. At this or at a later stage the patient has the right to be treated as a rational being. He should be encouraged to ask questions concerning his condition. It is an easy task as a rule to explain in simple language the significance of sensations which to him forbode all that is terrible because they are mysterious. I said quietly to one patient who was sullen and uncommunicative: "You will not go insane." It was a shot in the dark. His fervent "Thank God!" astonished me. We do not realize the vicious effect of suggestions ignorantly made by the patient's friends. It is they who create or confirm terrors in the victim's mind. As months go by, fixation of anxiety occurs, and the task of displacement becomes increasingly difficult. But the attempt should be made.

Treatment in hospital at an inconvenient distance from sympathetic friends is desirable in many cases, but the patient with mild symptoms should be encouraged to return to work as soon as possible. Only in this way can he quickly regain confidence in his own powers.

Medicinal treatment consists in the administration of potassium bromide and belladonna. Unfortunately the belief that bromide in average doses is a mental depressant dies hard. No drug is more effective in calming the mind and promoting refreshing sleep.

ACUTE UPPER ABDOMINAL PAIN.[1]

By J. COLVIN STORRY, M.B., Ch.M. (Sydney), F.R.C.S. (England),
Honorary Surgeon, Royal Prince Alfred Hospital.

THE subject of acute upper abdominal pain is of great importance alike to physician, surgeon and patient. Severe pain is unpleasant for the sufferer, but it has certain virtues; it is a danger signal to the patient and a guide to his medical adviser.

I wish to repeat the old old warning that no morphine should be given for the relief of pain until the surgeon has decided whether immediate operation is indicated. This point is made at the outset because the rule is broken too often. Valuable clinical signs may be masked by morphine.

There are many theories of the cause of pain in the abdomen, but as a surgeon I think of pain in terms of the disease it may connote. Experience has taught us that certain types of pain radiating in known directions and, accompanied by familiar symptoms and signs, usually indicate diseases which we have been accustomed to associate with such syndromes.

The question that I have in mind resolves itself into a differential diagnosis of such surgical conditions as are characterized by pain in the upper part of the abdomen. As you know, the umbilicus is normally just below the middle of the abdominal wall, and in some cases far below this level. Anatomists divide the abdomen into arbitrary regions by imaginary lines drawn vertically from the middle of each Poupart's ligament and horizontally to join the lowest point of each costal arch and the highest point of each iliac crest.

For our purpose a line drawn across the umbilicus may be considered to define the upper part of the abdomen, although, like a naughty boy, pain does not play always in his own "back yard", or should I have said front garden?

True abdominal pain may be divided into three classes: (i) The colic due to the spasmodic contraction of a hollow viscus or duct during its attempt to expel anything acting as an obstruction, (ii) the continuous pain of tension in similar organs, (iii) pain due to irritation of the peritoneum.

The abdominal wall is supplied by the lower seven thoracic nerves, the ilio-hypogastric, and the ilio-inguinal. The diaphragm, except the edges, is supplied by the phrenic nerve.

[1] Read at a meeting of the Royal Prince Alfred Hospital Residents' and Ex-Residents' Association, September, 1935.

I do not pretend to know the exact explanations of the unpleasant sensations experienced by sufferers from various abdominal crises, but have found an excellent guide in the work of Sir James Mackenzie and Dr. Head.

A. H. Burgess, of Manchester, in *The British Medical Journal* of December 11, 1920, published an admirable description of the signs and symptoms of acute abdominal crises, and I cannot do better than quote his paper. He stated that:

> Pain from the stomach is referred to the peripheral distribution of the fifth to eighth dorsal nerves, that is between the xiphoid cartilage and umbilicus, that from the duodenum close above the umbilicus, that from the small intestine and appendix (eighth to eleventh dorsal) to the umbilical area (the umbilicus corresponding to the tenth dorsal), and that from the large intestine to the mid-hypogastric line. From the liver, gall-bladder and bile ducts (seventh to ninth dorsal) pain is referred to the lower part of the epigastrium and also sometimes to the lower angle of the scapula (eighth dorsal).

When the peritoneum is irritated, it has been said that the pain is due to the noci-stimulation of the nerve endings of the extraperitoneal tissues and is felt just at the site of stimulation. In my opinion this view is correct.

To desert theory in favour of practice, I thought that it would be more interesting for my audience to discuss in turn the more important surgical emergencies that affect the upper part of the abdomen.

I have heard it said that the most important sign of death is that the patient "looks quite dead", and one may say that, as a rule, a sick man who requires the physician rather than the surgeon announces his requirement by his general appearance.

The worst pain of the upper part of the abdomen is probably associated with acute hæmorrhagic pancreatitis, and no less unbearable is that of perforation of a duodenal ulcer. Gall-stone colic can be almost as bad.

I have made a study of the records of perforated duodenal ulcer at this hospital from 1910 to June 30, 1935—a period of twenty-five and a half years—and of acute pancreatitis for the same period. It was considered that some facts would be more enlightening than any amount of theory.

Perforated Duodenal Ulcer.

Let us take the commoner disease first. Perforated duodenal ulcer is filed under the heading of "Duodenal Ulcer, Surgical". The work required the inspection of 817 envelopes and the perusal of 261 histories, the latter being the total number of perforated duodenal ulcers treated at the hospital in twenty-five and a half years. Five perforated jejunal ulcers are included in the series. Of the 261 patients, only 14 were females, so that in the experience of the hospital females suffer only in 5·36% of cases, and the incidence is, on the average, ten *per annum*—less than most people would expect. In Table I the cases are set out according to age of patients in decades, sex and mortality rate. The findings are shown graphically in Figure I. You will notice that the chance of survival decreases with age, and that seven of the 14 females died. The law of chance was illustrated over and over again while searching the records. Although the disease is so rare in

TABLE I.
Showing Age in Decades.

Decade.	Males.	Females.	Deaths.	Mortality Rate.
2	3	0	0	—
3	31	2	4	12·12%
4	72	1	11	15·06%
5	72	3	13	17·33%
6	51	5	14	23·21%
7	13	1	6	42·85%
8	3	2	4	80·0%
9	2	0	2	100·0%
Totals ..	247	14	54	20·69%

Excluding Number 555 (arrived dead), and Number 810 (died half an hour after admission), the deaths were 52, and mortality rate was 20·07%.
Females: Total 14; deaths, 7; mortality rate, 50·0%.
Ages of patients who died: 41, 47, 54, 55, 64, 73, 79.

females, at one stage three successive patients were women, and they all came under my care. There were 54 deaths in the series, giving a gross mortality rate of 20·69%. If Number 555, who was dead on arrival at the ward, and Number 810, who died half an hour after arrival, are excluded, the rate is 20·07%. The youngest patient was eighteen, and the oldest eighty-seven years of age.

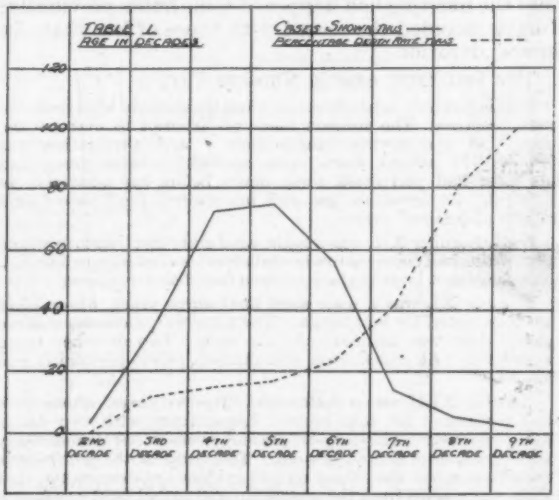

FIGURE I.

Now if you will look at Table II, the time that had elapsed between perforation and admission will be seen. The findings are shown graphically in Figure II. Of the 114 patients who were admitted to hospital less than six hours after perforation only seven died,

TABLE II.
Duodenal Ulcer. Showing Time that Elapsed between Perforation and Admission to Hospital.

Period of Time.	Patients.	Deaths.	Mortality Rate.
No information	13	3	23·0%
Less than 6 hours	114	7	6·14%
6 to 12 hours	58	11	18·96%
12 to 18 hours	21	5	23·8%
18 to 24 hours	7	6	85·7%
One to two days	23	10	43·5%
Two or more days	17	10	58·8%
One week or more	8	2	25·0%

making a mortality rate of 6·14%. It may be assumed that approximately two hours would as a rule elapse between admission and operation, and that the prognosis would be good if the patients were operated on less than eight hours after perforation.

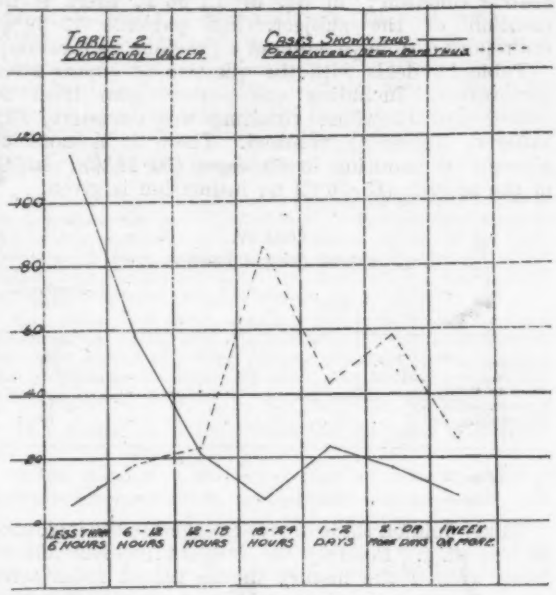

FIGURE II.

You will notice that the death rate rises steeply as the interval between the catastrophe and treatment increases, until after the lapse of twenty-four hours it falls again because some of these perforations become sealed off by nature. This natural form of defence had been made in some of the patients on whom I operated. I have seen, occasionally, a man who had been ill for more than a day, and who could be suspected reasonably of having had perforation of a viscus, with all signs of urgency disappearing. Such a patient has been left to recover. Masterly inactivity should be practised in very rare circumstances, but when events justify such behaviour, the surgeon must have the courage to do nothing.

In thirteen of the histories there is nothing to indicate when bursting of the ulcer had taken place.

TABLE III.
Showing History of Previous Indigestion.

Period.	Number of Cases.
Four weeks or less	27
More than four weeks	128
Indefinite—time not stated	42
Total with indigestion[1]	197
No indigestion	30
No information	34
Grand total	261

[1] These cases include six with previous perforation.

Table III shows the incidence of symptoms of previous indigestion. One hundred and ninety-seven patients, or 75·47%, made a definite statement of antecedent dyspepsia. In 30 cases, or 11·49%, there is a note of lack of previous interference with gastric function; in 34, or 13·06%, there is no mention of the subject. Six patients (2·29%) are reported to have suffered a previous perforation.

Table IV deals with the question of emesis after perforation. Including one patient who tried to vomit, and 17 whose vomiting was excessive, 141 sufferers (54·02%) vomited. There is a note of absence of vomiting in 79 cases (30·26%); in 41 of the records (15·70%) no intimation is given.

TABLE IV.

Showing Degree of Vomiting.

Type.	Number of Cases.
Vomiting +	123
Vomiting ++	17
Tried to vomit	1
No information	41
No vomiting (−)	79
Total	261

In my opinion these figures exaggerate the incidence of vomiting. Possibly the resident medical officers have written the history in the record room some days after the operation, and have been so accustomed to associate abdominal pain with vomiting that they have automatically recorded vomiting whether it had taken place or not. I have always regarded vomiting as rare after the perforation of a duodenal ulcer. True, it does occur, but not nearly so regularly as in the case of persons suffering from appendicitis or gall-stone colic.

Table V gives the occupations of the patients. No conclusion can be drawn from this set of figures. For some reason the condition was very rare among soldiers on active service, yet in this series, of the cases amongst males 70·84% occurred in men between the ages of twenty and forty-nine. The majority of members of the Australian Imperial Force must have belonged to this age group. I think that the regular open air life must have been a factor in prevention. I should like to hear from some of the men who practise in the country whether the disease affects men who work in the open.

TABLE V.

Occupations.	Number of Cases.
Labourers	66
Domestic duties	10
Old age pensioners	9
Carters	8

Twenty-eight perforations occurred while the patients were at work, one occurred after the patient had been rowing, and one patient is said to have been struck while he was at work, so that 30 perforations (11·49%) were associated with work in point of time at least. Fourteen (5·36%) ulcers perforated while the patients were asleep, and the patients were awakened by the pain.

The question of aggravation of a preexisting duodenal ulcer by work is a contentious subject nowadays. Workers have won awards before the Workers' Compensation Commission on this point. Who knows whether exertion can precipitate a perforation? It must be admitted that bodily movement takes place even during sleep, and that an ulcer perforates without the patient's indulging in exertion. However, what man among you, knowing that he had an ulcer covered by a thin film of peritoneum, would fail to keep as quiet as possible?

It is my impression that many of these patients suffer a perforation soon (up to an hour) after the ingestion of solid or fluid food. Upon this subject I could find little information. There is no mention in 237 cases; positive information is given in 22, and negative information in 12, including one in which the disaster overtook the patient two hours after Christmas dinner.

Let us consider the patients who died and who had been admitted to hospital less than six hours after perforation. When the context of the notes indicated that the bursting had happened some hours previously, I have included the record with those of less than six hours' duration.

The first fatal case is Number VII.

In this instance appendicectomy and laparotomy with drainage were practised. The surgeon made no attempt to oversew the ulcer. At *post mortem* examination a large perforation was found. The patient was a male, aged thirty-seven years, and his ulcer had perforated some hours before his admission to hospital. At operation gas and bile-stained fluid were found in the abdominal cavity.

Next, Number XII, was a male, aged sixty-five years, in whom perforation had occurred five hours before his admission to hospital. At operation a large perforation was found and oversewn.

Number XL was a male aged forty-three years, whose ulcer had perforated for four hours. The ulcer was oversewn and the gall-bladder was sutured over the area. Two drainage tubes were used. At *post mortem* examination pelvic peritonitis was found.

Number XLII was a male, aged fifty-five years, whose ulcer had perforated for four hours. Laparotomy with two drains was performed. The surgeon did not oversew nor deal actively with the hole in the duodenum. The notes of the *post mortem* examination say that there was no evidence of oversewing, that there was a sinus from the operation, and turbid fluid was found, mostly in the pelvis.

Number XLV was a male, aged fifty-two years, whose ulcer had perforated for three hours. The patient had been a devout worshipper at the shrine of Bacchus. At operation a large perforation was found and oversewn and a pelvic drain was employed. At the *post mortem* examination bronchopneumonia and chronic nephritis were demonstrated. There was no general peritonitis.

Number XLVI was a male, aged forty-four years, whose ulcer had perforated for five and a half hours. The ulcer was oversewn. *Post mortem* it was found that the sutures had pulled away.

Number XLVIII was a male, aged fifty-five years, whose ulcer had perforated one hour before his admission to hospital. The ulcer was oversewn. *Post mortem* no explanation of the cause of death was found.

It may be assumed that patients Numbers VII and XLII died because the surgeon did not attempt to close the perforation. In the notes there is a third patient whose ulcer had perforated seven hours and who was treated in the same way with the same result.

In the case of patient Number XII, old age was the lethal factor. No reason is apparent for the death of Numbers XL and XLVIII. Alcohol and bronchopneumonia seem to have been the actual causes of the death of Number XLV.

In the case of Number XLVI the sutures carried away. Fatal case Number XXXIII is shown in the one to two days after perforation series.

The patient was a female, aged sixty-four years, who had had epigastric pain for two months and vomiting for five days and pyuria. She consulted a urologist, who sent her to hospital for cystoscopic examination. After this procedure the ulcer perforated, apparently during the night while she was in hospital, and the perforation was discovered at *post mortem* examination. Morphine had been given to relieve her pain.

Table VI gives the provisional diagnoses. The provisional diagnosis seems to have been a doubtful quantity. Sometimes it was the preoperative diagnosis of the surgeon, and at other times the opinion of the doctor who sent the patient to hospital. On some occasions it has been written perfunctorily by the resident medical officer some time after operation. However, when the diagnosis has been "perforated ulcer" and the surgeon has begun with an incision over the appendix, I have altered the preliminary diagnosis to "appendicitis".

TABLE VI.
Showing Provisional Diagnoses.

Diagnosis.	Number of Cases.
Perforated ulcer	202
Appendicitis	21
Acute abdomen	11
Intestinal obstruction	9
Cholelithiasis	7
No information	5
Lead colic	1
Cancer of stomach	1
Hepatic abscess	1
Pyuria	1
Strangulated hernia	1
Double pneumonia	1
Total	261

In the records there is reasonable evidence to say that in 202 of the 261 cases (that is in 77·39%), the preoperation opinion was correct. The mistake of calling the condition appendicitis was made on 21 occasions—an error of 8·04%.

Diagnosis is easy in a typical case. Of the patients 94·64% were males, and of these 70% were between the ages of twenty and forty-nine.

The history should be carefully taken. Almost invariably the story will be of sudden severe and unbearable pain. It is felt as a rule high up and to the right, although some of the patients have described it as being all over the abdomen, and others have said that the initial pain was felt about the umbilicus and even lower. In my own experience food has often been ingested less than an hour before the onset, and vomiting is rare, and certainly not persistent till the late stages. Sometimes pain is present in the right shoulder from irritation of the phrenic nerve.

The appearance is characteristic, and with the history one look at the patient is enough. He lies in bed afraid to breathe or to move. Respiration is short, grunting and thoracic. The abdomen is as hard as a board. An occasional patient has taken castor oil. Many, like Cassius, have "a lean and hungry look". When the initial pain has been felt low in the abdomen, probably the hole has been big and a shower of acid chyme has fallen at once.

The perforation may be pin-point or as large as a threepenny piece. The stage of "dangerous improvement", described by Dr. William Mayo, must be borne in mind, as some of the patients have said that they had felt better after about an hour.

Now let me tell two stories.

Some years ago I was asked by a resident medical officer to see a woman who had "gall-stones". She had tenderness in the right hypochondrium and had pain at the tip of the right shoulder. I made an incision over the gall-bladder and blood gushed from the peritoneal cavity. A second mid-line incision disclosed a ruptured ectopic gestation.

Also, some years ago a certain gynæcologist rang me on the telephone just before midnight and said that he was sending to hospital a woman who had a perforated gastric ulcer. I replied that a colleague was doing my work after midnight. The said colleague calmly removed a ruptured ectopic gestation.

The moral is that if you fail to take a careful history "it serves you right".

When patients are seen a day or two, or even a shorter interval after perforation has occurred, all the signs may be in the lower part of the abdomen. In the same way blood from an ectopic gestation may run quickly to the upper part of the abdomen and trick the unwary.

In regard to treatment I consider that it is wise to do only as much as is necessary to save life. Open the abdomen by a vertical incision through the rectus about 5·0 centimetres (two inches) to the right of the mid-line. This will bring you almost invariably straight to the ulcer. There is always much induration, and it is impossible to use a purse-string suture. It is best to make a mattress suture wide of the hole, and safest to pass each end of the thread from above downwards. (See Figures III and IV.)

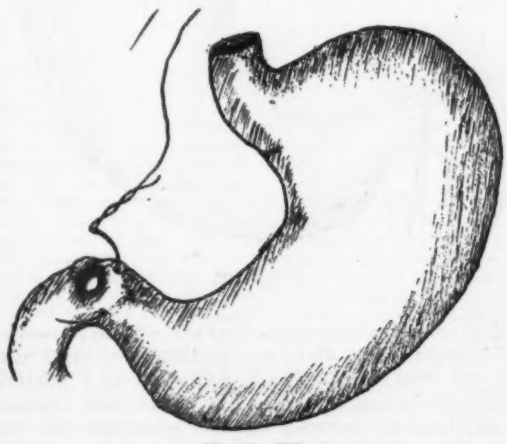

FIGURE III.

Tie this and, leaving the ends long, use it as a guide to steady the duodenum. (Figure V.) Now tie a Lembert suture or another mattress suture above

and below and a third over the original mattress suture. (Figures VI and VII.) Finally, suture with catgut a convenient tag of peritoneal fat over the line. (Figure VIII.) I prefer to use silk for the bowel threads. In the records there are notes in the accounts of two *post mortem* examinations, showing that the sutures had failed to hold. Of course these

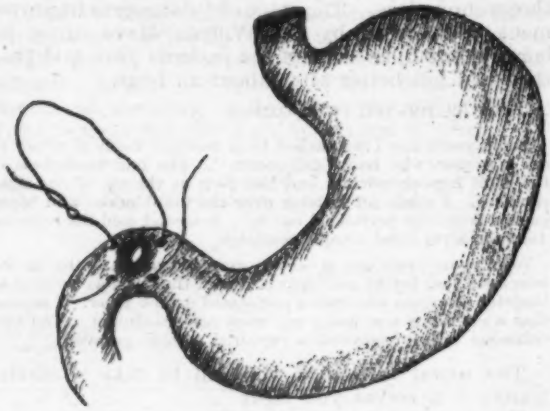

FIGURE IV.

accidents may, or may not, have been due to absorption of catgut. I have generally used silk, and silk that is not too fine, and have not seen harm come from this practice. I do not use a drain unless the condition is old and pus is present. The local chyme is sucked out with the suction apparatus and the peritoneum is assaulted as little as possible.

FIGURE V.

Most of my colleagues favour the simple procedure, but in fairness to the more radical method I must tell you that one surgeon who performs posterior gastro-enterostomy almost as a routine measure in addition to oversewing has obtained the best immediate results. Nevertheless, no surgeon in a lifetime deals with enough cases to draw any logical conclusion, and it is sound surgery, in the presence of an abdominal emergency, to be deliberate and quick, yet neither meddlesome nor hasty.

The prognosis depends on the age of the patient, the duration and size of the perforation, and on the judgement and skill of the surgeon.

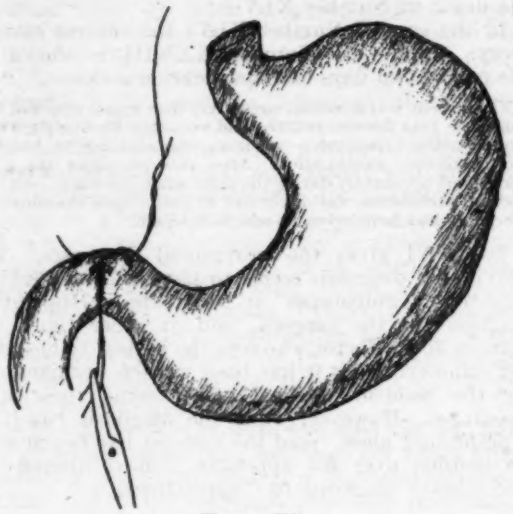

FIGURE VI.

Acute Pancreatitis.

The figures of acute pancreatitis only reveal our ignorance of the subject.

In twenty-five and a half years there have been only 29 cases of genuine acute pancreatitis. There were 18 female and 11 male patients. The youngest patient was aged twenty-four, and the oldest seventy-

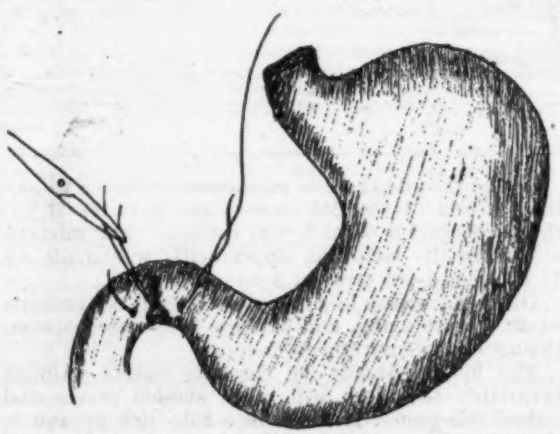

FIGURE VII.

seven. Table VII shows the age in decades and Table VIII the diagnosis. The findings are shown graphically in Figure IX. You will note that only two cases are reported to have been correctly diagnosed, and that 22 patients died. The mortality rate is 75·86%.

Most of the patients had been ill for one to two days before admission to hospital. Repeated vomiting was noted in 22 instances, and vomiting in five.

TABLE VII.

Showing Age in Decades of Patients with Acute Pancreatitis.

Decade.	Number of Patients.	Number of Deaths.	Mortality Rate.
3	7	3	42·85%
4	7	7	100·0%
5	3	1	33·33%
6	4	3	75·0%
7	5	5	100·0%
8	3	3	100·0%
Total	29	22	75·86%

TABLE VIII.

Showing Diagnoses in Cases of Acute Pancreatitis.

Diagnosis.	Number of Cases.
Intestinal obstruction	10
Cholecystitis	7
Acute abdomen	2
Perforated ulcer	2
Acute pancreatitis	2
Peritonitis	1
Appendicitis	1
Diabetic coma	1
No information	3
Total	29

Gall-stones were found in seven patients.

In only seven patients were gall-stones found at operation or at *post mortem* examination. There are 113 cases of pancreatitis of all degrees filed in the records for the twenty-five and a half years. There is one only as a complication of mumps. You see that the biggest error in diagnosis is that of calling the condition intestinal obstruction. The reason

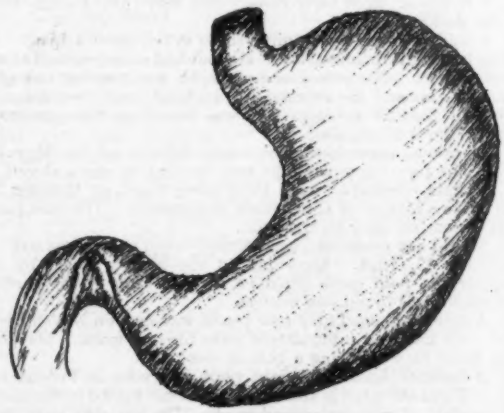

FIGURE VIII.

probably is that the patients have a virtual obstruction high in the alimentary canal because of interference with the nerve supply of the duodenum.

So far as I know, I have treated only five patients with acute pancreatitis, three of this series and two at the Coast Hospital.

Of the former, one was a male aged seventy-two years, who was deaf and nearly blind, and who had been ill for two days with pain in the epigastrium and both hypochondria. He had vomited frequently. Laparotomy with drainage was performed, bile-stained fluid and fat necrosis being found. At *post mortem* examination acute hæmorrhagic pancreatitis and gall-stones were demonstrated.

The next patient was a female, aged twenty-four years, described as being very fat. She had been ill for two days with severe epigastric pain. Vomiting had occurred. There was no rigidity. I made an incision over the appendix and found blood-stained fluid and fat necrosis. Cholecystostomy was performed, forty gall-stones being removed. The peritoneum was drained. At a subsequent operation the gall-bladder was anastomosed with the jejunum. This patient reported to me in July, 1935—nine years after the first operation—quite well except for an incisional hernia.

The third patient was a male, aged fifty-seven years, whose gall-bladder had been drained three months previously. He had been ill for one day with sudden agonizing pain in the left side and in the right shoulder blade. Vomiting had been excessive. I made an appendiceal incision and found bile-stained fluid and then drained the abdomen through the old wound. At *post mortem* examination acute gangrenous pancreatitis was described.

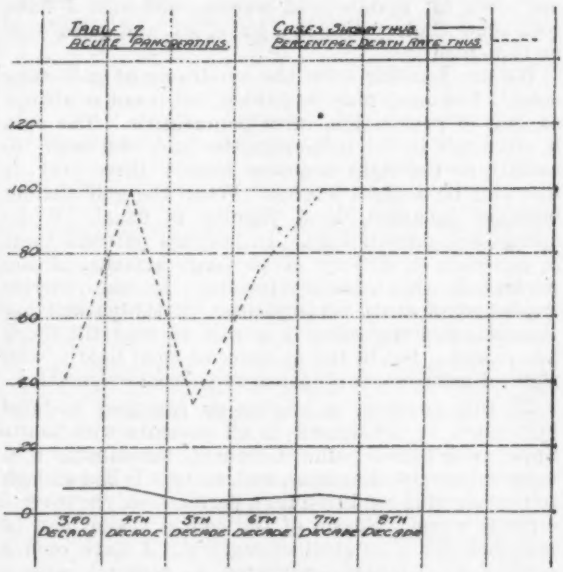

FIGURE IX.

Of the other two patients the first was a young man who had been kicked in the abdomen two days before his admission to hospital. He was in great pain and had a very rigid abdominal wall. Diffuse fat necrosis and blood-stained fluid were found. I put a tube into the pancreas and the lad recovered.

The second was a fat returned soldier who had lost a leg at the War. He had had indigestion for some weeks. He went into town, bought some fruit and ate one banana, which he vomited at once. Soon after this he got terrific pain in the epigastrium, more on the left side. He collapsed at once, and as he said "fell" into a taxi and was so ill that the driver called at the house of the local doctor. I saw him in hospital within twelve hours. He was utterly collapsed, and had a very rigid abdominal wall. He had not vomited again. He had extreme abdominal tenderness, especially towards the left loin. I thought he had a perforation of the stomach. Just as he was going under the anæsthetic he vomited dark brown fluid. I remarked that this was against a diagnosis of perforation. His abdomen was full of dark fluid, with some blood clot round the head of the pancreas. I drained the peritoneal cavity. The patient died less than an hour after operation. *Post mortem* the whole pancreas was found to resemble one long blood clot.

Just as I was wondering whether operation was justified in these patients I found, in *The Australian and New Zealand Journal of Surgery* of July, 1935,

a review of an article of O. Mikkelsen, of Copenhagen. This surgeon deprecates any primary operation, and admits diagnosis as the major difficulty. He mentions absence of rigidity as one point of a syndrome which should make diagnosis possible. I cannot agree, as some of the patients have had rigidity. He also says that estimation of the urinary diastase is a valuable supplement to clinical methods. I should like to hear what the bio-chemists say about this.

It seems that a very sick patient who has had severe upper abdominal pain with unusual signs may have pancreatitis. You will notice that acute pancreatitis occurs more often in females than in males in the ratio of 18 to 11.

Gall-Stone Colic and Cholecystitis.

Our next consideration is gall-stone colic and acute cholecystitis. The subjects of these maladies are often fat middle-aged women, although I have seen acute cholecystitis in a girl of six, and gall-stones in thin men.

We are familiar with the syndrome of gall-stone colic. The onset may be sudden, but is not so abrupt as that of perforation, or of pancreatitis. The pain is often felt in the mid-epigastric line, and tends to radiate to the right shoulder blade; there may or may not be a slight icterus. When the gall-bladder becomes inflamed, local rigidity is usual. Widespread tenseness is rare. In hospital patients there is generally a history of previous attacks. I am persuaded that cholecystostomy is the correct treatment of acute cholecystitis. If the patient has typical gall-stone colic, it is wise to wait till shock has passed; but in the presence of local rigidity and signs of toxæmia, it is foolish to postpone operation.

In this country, a leaking or ruptured hydatid cyst must be considered in all patients with acute upper abdominal pain. Coronary thrombosis is a very fashionable diagnosis, and, while it is bad enough to mistake this condition for a perforation, the reverse error is worse. Spasm of the cœliac vessels can be mistaken for a surgical emergency. I have seen a patient with tetanus admitted to hospital with a diagnosis of an abdominal crisis. Renal colic is sometimes projected forwards, but as a rule no confusion need arise. Perforation of a gastric carcinoma is rare, as is the bursting of the gall-bladder. Malaria may mimic any disease. And now, last, but not least, acute appendicitis is the most common cause by far of all abdominal emergencies, and the initial pain is often high, so that it may be difficult to exclude the old favourite.

And this is the conclusion of the whole matter: when you are called to a patient who is overwhelmed with upper abdominal pain, act promptly, for delay may mean death.

Acknowledgement.

I wish to thank Miss K. M. Cummins, who is responsible for the drawings and the graphs appearing with this paper

Addendum 1.

After reading this paper I examined carefully the records of the thirty cases of perforated ulcer in which the condition was associated with work. In 15 there is only a note in the history that the pain came while the patient was at work. The occupations of these sufferers is given as: labourers, 5; wharf-labourer, 1; carpenters, 2; fireman, 1; storeman, 1; fitter's examiner, 1; printer, 1; ironworker, 1; brassmoulder, 1; and doorkeeper, 1.

In the second group there are eight cases

1. A labourer, aged twenty-one years, had had pain after food for fifteen days. He had rushed out one morning without eating and had rowed for over an hour. The pain had come after this exertion.

2. A painter, aged forty years, had had a "gastric ulcer" for six years. He had been sent to hospital by the medical officer of Number 4 Australian General Hospital (Randwick). The onset of the pain had been ten minutes after "holding a patient".

3. A fisherman, aged thirty-seven years, had for twelve months had pain a quarter of an hour after food. The pain had come while he was fishing.

4. A wharf-labourer, aged thirty years, had had "gastritis" three months ago. He had been struck while working, the day before his admission to hospital. On rising on the day of admission he had had a glass of lager beer. He had had severe pain at 6.30 a.m.

5. A textile-worker, aged thirty-two years, had had indigestion for two years. The pain had come "just as he was knocking off work and drinking cold tea".

6. A rigger, aged forty-six years, had pain after food for six months. He was a heavy drinker. Three days ago, while chopping wood, he got a sudden pain. At 9 a.m. (on the day of his admission to hospital), shortly after having a drink, he got severe pain and had another drink, and the pain doubled him up.

7. A machinist, aged thirty-six years, was operated on for duodenal ulcer six years ago. He was well till 9 a.m. on April 1, 1935, when he had severe generalized abdominal pains. No operation was performed. No *post mortem* examination was made. A certificate was given by an outside doctor.

8. A blacksmith, aged thirty-two years, had severe pain four days ago all over the abdomen while at work. He had had pain one-half to two hours after food for eleven months. No note was made of perforation having been seen at operation. The findings indicated perforation.

In the remaining seven cases there is a more definite statement of association with effort, having regard to the apparent time of perforation.

1. A milkman, aged forty-three years, had a perforation while he was milking.

2. A milk vendor, aged thirty-three years, gave a history of having had eight drinks a day. He had had an occasional touch of indigestion. The pain came while he was running out of a customer's house. He worked for an hour, took two doses of oil and a Seidlitz powder. Oil was found in the peritoneal cavity. The patient died.

3. A groom, aged forty-five years, became ill on May 25, 1927. The pain came while he was bending to use a shovel.

4. A tram conductor, aged forty-three years, on October 25, 1929, gave a history of three years' indigestion. The pain came while he was on duty on the tram.

5. An old age pensioner, aged eighty-seven years, was seen on November 17, 1934. According to the history given by his daughter the pain came while he was working in the garden. Perforation was found *post mortem*.

6. A storeman, aged fifty-four years, was seen on November 3, 1934. He had had intermittent pain for one week. The pain came while he was lifting a bale of wool.

7. A barman, aged forty-eight years, was seen on February 3, 1915. This case is not in the series, but is mentioned in discussing the question of perforation and work. The patient's pain came while he was lifting a cask. *Post mortem* examination revealed a perforation on the anterior surface of the stomach near the lesser curvature. On cutting the ulcer the pathologist considered that it "looked malignant".

Addendum 2.

A male patient, aged forty-five years, a taxi-driver, was admitted to the Royal Prince Alfred Hospital on October 31, 1935. This man had suffered a perforation of a duodenal ulcer in October, 1922, and the perforation had been oversewn by me. The patient stated that since his last illness he had suffered

some discomfort at intervals, usually of three months. On the morning of his admission to hospital he had had his breakfast at 9 a.m. At 10.30 a.m. he had drunk some "horehound" beer. Soon after this he had taken an antacid powder with water. At 11 a.m. he was sitting "doing nothing" in his Ford motor car, letting the engine warm, when he had sudden agonizing pain all over the abdomen. He was operated on by me at 5 p.m., and a large perforation of the duodenum was found. The hole admitted easily the end of the nozzle of the suction apparatus. The theorists may choose as the causative factor in the perforation of this patient's ulcer a breakfast of cereals, a glass of "horehound" beer, an antacid powder, or a Ford motor car.

On October 28, three days before this patient's ulcer perforated, his wife received a letter from a friend in the country whose husband had had a perforated ulcer eighteen months previously. This woman had also given her husband "horehound" beer, and, strangely enough, he suffered from a second perforation of his duodenum on October 28. Nothing would convince these two women that their husbands' illnesses were not due to the "horehound" beer.

Reports of Cases.

LOCAL AND GENERAL ANAPHYLAXIS.

By ROBERT S. STEEL, M.B., Ch.M. (Sydney), M.R.C.P. (London),
Clinical Assistant to Medical Out-Patient Department, Royal Prince Alfred Hospital, Sydney,

AND

DAVID ADCOCK, M.B., Ch.M. (Sydney),
Sydney.

A MALE patient contracted a streptococcal infection of the throat, with blebs on the fauces and œdema of the uvula. *Streptococcus hæmolyticus* was obtained on culture. The œdema spread down the trachea and the patient died of œdema of the glottis and trachea, although a tracheotomy was performed. This occurred on February 7, 1935, and the duration of his illness was two days.

Dr. X, a man of abnormally strong physique, who attended the patient, developed a sore throat twenty-four hours later. On examination there was a marked redness of the fauces, and blebs were present on the soft palate. There was some œdema of the uvula. *Streptococcus hæmolyticus* was isolated for the preparation of a vaccine.

On February 13 thirty cubic centimetres of polyvalent antistreptococcal serum were given intramuscularly into the left arm and ten cubic centimetres of antistreptococcal (stock) vaccine subcutaneously. On each of the following two days thirty cubic centimetres of serum were again given intramuscularly in the left arm. The blebs on the fauces cleared up and there was less congestion, but Dr. X experienced the aches and pains and lethargy which we associate with influenza, although the temperature was normal.

On February 18 there was a patch of erythema on the skin of the arm, about 10·0 centimetres (four inches) in diameter, over the site of each of the three injections. Next day there was marked generalized urticaria, which was relieved to a slight extent by adrenaline. The following day Dr. X developed pains in the region of the right wrist and left elbow.

On February 21 a subcutaneous injection of fifty million organisms of autogenous vaccine was given. The following day the temperature rose to 37·4° C. (99·4° F.), and there were pains round the elbows, knees, wrists and fingers. There was no noticeable swelling of the joints and no fluid could be detected. The pains were very acute, and Dr. X was confined to bed. Not constant in position, the pains would move from one side of the joint to the other, or be felt more in another joint region, and movement was agonizing. Dr. X affirmed that the pains were not in the joint, but felt as though they were in the tendons and capsule of the joints.

On February 24 the urticaria had subsided. The local serum reactions on the left arm were fading, but were still present, and the whole of the left arm was painful. The pains around the joints were excruciating, the temperature was 38·7° C. (101·8° F.), and the heart rate was rapid and out of all proportion to the temperature. There was intense prostration. The colour was a grey pallor. The systolic blood pressure was 110 and the diastolic pressure 60 millimetres of mercury.

Adrenaline, 0·3 cubic centimetre, was given with no effect on the pains, and the systolic blood pressure fell immediately to 100 millimetres of mercury. In half an hour the systolic reading was again 110 millimetres and the dose of adrenaline was repeated with similar results. Pituitrin, 0·5 cubic centimetre, was given and repeated every two hours with marked relief of pains.

On February 26 the temperature became normal and pains had gone. The pituitrin was discontinued.

Except for a slight rise to 37·6° C. (99·8° F.) next day, the temperature has remained normal. There have been some aching pains in the shoulders since.

During this illness Dr. X lost over 6·3 kilograms (one stone) in weight.

Comment.

Points of interest in this case are:

1. The careful observation by a physician of an unusual illness which occurred to himself.

2. A local allergic reaction at the site of the serum injections, although there is no history of previous serum injected, and no family or personal history suggesting a latent allergic condition.

3. Severe pains which were experienced in the structures surrounding the joints and not in the joints themselves.

4. The tachycardia, intense prostration and loss of weight.

5. The action of adrenaline, which caused a fall in blood pressure, and its inability to relieve the pains, which we believe to be due to tension caused by urticarial œdema in the joint capsules.

6. The beneficial use of pituitrin in repeated doses.

It is reported that arthritis occurs as a manifestation of "serum sickness" in about 1% of cases. This arthritis is not described at any length in the literature. It would appear that it is in the nature of an internal urticaria and the joints are not affected, the urticaria being present in the periarticular structures. Our experience has taught us in more cases than one that adrenaline does not always relieve generalized urticaria. Pituitrin is most useful in some of these cases, just as it has been in the localized urticaria (we believe) of Dr. X.

Reviews.

DISEASES OF THE NERVOUS SYSTEM.

WE have before us the sixth edition of the now well known text-book of "Diseases of the Nervous System", by Jelliffe and White, of New York, the reappearance of which makes it plain that appreciation by the profession has not been wanting.[1] In form the book remains unchanged, but it has been thoroughly revised and in parts added to or rewritten. To those unacquainted with the volume we may say that it offers an account of nervous and mental diseases viewed from every level of

[1] "Diseases of the Nervous System: A Text-Book of Neurology and Psychiatry", by S. E. Jelliffe, M.D., Ph.D., and W. A. White, M.D.; Sixth Edition; 1935. Philadelphia: Lea and Febiger. Royal 8vo, pp. 1175, with 497 engravings and 13 plates.

activity. Vegetative or visceral neurology, the endocrinopathies, sensori-motor neurology (affections of brain, spinal cord and peripheral nerves), the neuroses, psychoneuroses and psychoses are all discussed. Thus it will be seen that as a text-book it is unusually comprehensive and ambitious; however, in their preface the writers explain their ideal by saying that "a medical specialty is but one aspect of medicine in general, and in order that the specialty may adequately be understood it must be formulated in terms of the fundamental principles of the larger whole of which it is a part".

It must be obvious that to deal satisfactorily with all these subjects under one cover is a heavy task, and frequently the authors demonstrate their handicap by referring the reader to other publications when he is worked up to expectation of a fruitful dissertation. Suggestively it occurs to us that pages might be gained and used for amplifying material descriptions by omitting schemes of clinical examination as well as facts of physiology and anatomy which are to be found in specific manuals and in which the advanced student, for whom we judge this book to be intended, is, or ought to be, already grounded.

None will deny that visceral neurology and endocrinology are subjects of the highest importance; at the same time our knowledge of them is far from mature, and in this book we notice a tendency, not consonant with the principles of text-book writing, to set forth as proved points concerning which there is by no means unanimity. Also we may mention that in these chapters, and indeed throughout the book, the reader who has not mastered Freud's difficult dialect, will find passages outside his comprehension; moreover, he may see the advantages of psychoanalysis pressed in unexpected places.

Notwithstanding that the wide scope of the undertaking almost precludes equality, taken as a whole, the descriptive writing is clear, succinct and reliable; in fact the descriptions of some diseases approach perfection. Allusions to the remarks of bygone observers and writers set down at the head of many descriptions are most refreshing, as well as indicative of wide reading; so likewise are the free quotations from important and significant literature, the source of which, by the way, is conveniently indicated by footnotes, instead of by a terminal and perhaps unread bibliography. Lastly, the illustrations could hardly be improved upon and the printing is excellent.

VARICOSE VEINS AND HÆMORRHOIDS.

"THE CURE OF HÆMORRHOIDS AND VARICOSE VEINS", by Stuart McAusland, is a small book of seventy-four pages, which has already, in two years, reached its second edition.[1]

The main bulk of the work is taken up in dealing with the diagnosis and treatment of hæmorrhoids and varicose veins, but chapters are also included dealing with the modern ambulatory treatment by bandaging of varicose veins. There is also a chapter on the injection treatment of other conditions, such as hydrocele, varicocele, and ganglion. With regard to the last mentioned the directions are clear and the claims are moderately stated.

The book is well illustrated with actual case photographs and also with diagrams illustrating technique. The various injection fluids for both varicose veins and hæmorrhoids are discussed and the results are compared. The author favours at present for hæmorrhoids 5% carbolic acid in almond oil, and for varicose veins a new solution with lithium salicylate in 35% solution with 1% "Ethocaine", which he declares is more suitable for ordinary cases than quinine solutions.

The author, who, by the way, is a physician and not a surgeon, has had an extensive experience in charge of a clinic in Liverpool, England. His many tips are useful and practical and his directions are clear and concise. One can find no fault with the technique that he describes; it has by now become practically standardized. Altogether this is a useful book for the general practitioner or out-patient surgeon, and especially for those new to this class of work.

LIVING THINGS.

THERE is at present what amounts almost to a sudden "eruption" of text-books of elementary biology by English authors. No doubt this reflects the fact that at long last the educational authorities in England have recognized how much they have neglected biology and how much out of step they were with such countries as the United States of America, Germany, France and Japan in this respect. It is obvious that biology as a school subject is at last coming into its own in Great Britain.

Unfortunately most of the authors cannot rid themselves of the shackles of the past, and their conception of biology is merely the conjunction of a little zoology and a little botany leavened by some physiology. It is a great relief, therefore, to come upon an elementary text-book like that of Richard Palmer, with the title "Living Things: An Introduction to Biology".[1]

The author happens to be a lecturer in education in a university where the department of education has always kept a cultural training prominently before the miscellaneous students entering its class rooms. This may explain his outlook, which is unfortunately unusual, although it really ought to be universal.

As a guide to the young student the book is excellent. It is readable too. In the first part the fundamental phenomena characteristic of all living organisms are considered. The chapters deal with cells and organs, animal nutrition, plant nutrition, respiration in the two kingdoms, transport in animals and the significance of water and the green plant, responses to stimuli in regard to both plants and animals, and finally to reproduction. The living world is thus regarded as a whole. Simple experiments are suggested and instructions are given for carrying them out.

The second part of the book deals with the diversity of living things, and a suitable selection of different animal and plant types is described as examples. The book concludes by discussing the relationship of living organisms to the environment, giving a brief account of evolution and heredity, and finally, and most unusual of all, discussing the methods of science.

Australia is a long way behind England in regard to its school science, and biology rarely appears as a subject in our curricula. Actually it is, if anything, a subject the introduction of which one would have expected in Australia before England—but we are great plagiarists. Biology will, however, we understand, appear in the New South Wales secondary school syllabus in the very near future, and so probably a chance will be given, not only to intending medical students, but also to the far greater number of children who are never to reach our universities at all, and who now go out into life knowing little or nothing of the science which concerns humanity most of all.

It will be difficult no doubt to introduce the subject into some boys' schools, where a narrow outlook may take some shifting. Unfortunately education is still too much a trade, with vested interests. The eventual outcome is, however, fairly certain.

Until the time arrives when a special text-book of biology can be written for the Australian boy and girl, this one may prove very useful indeed. It is probably the best elementary text-book we have handled. It is well illustrated and has a good index.

[1] "The Cure of Hæmorrhoids and Varicose Veins", by S. McAusland, B.A., M.D., Ch.B., M.R.C.S., L.R.C.P.; 1934. London: John Bale, Sons and Danielsson, Limited. Demy 8vo, pp. 85, with illustrations. Price: 4s. net.

[1] "Living Things: An Introduction to Biology", by R. Palmer; 1935. London: George Allen and Unwin, Limited. Crown 8vo, pp. 400, with illustrations. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, JANUARY 11, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

OCCUPATIONAL THERAPY AND ITS MODERN DEVELOPMENT.

NOT nearly enough attention is directed in Australia to occupational therapy. When a person is stricken with physical or mental illness a diagnosis of the condition is made, the medical attendant prescribes certain remedies, a nurse or masseur may carry out instructions given by the medical attendant and Nature does the rest. The patient is seldom called upon to exert himself in a way that will contribute directly to his cure. Occupational therapy has been defined as any activity, mental or physical, prescribed and guided for the purpose of contributing to or hastening recovery from injury or disease. The subject has been discussed in these pages on previous occasions. The present object is, in the first place, to bring the subject of occupational therapy again before medical practitioners so that they may not forget its possible application in private and particularly in hospital practice; in the second place, to point out that occupational therapy cannot be successfully organized without the employment of people specially trained to apply it (they are usually known as aides); and in the third place, to draw attention to what may be regarded as an extension of occupational therapy recently reported from America.

Occupational therapy is not always necessary. Illnesses sometimes arise in which the best form of occupational therapy is its avoidance. A broken leg, an acute appendicitis, a strangulated hernia may come as a godsend to a harassed and busy man who imagines that the whole of his little world will come toppling about his ears if he stops working for an instant. Occupational therapy is required chiefly in long and chronic illnesses; its most useful sphere is in mental conditions. The chief object may be to create a diversion, to take the patient's attention from his illness, and to give him courage and confidence. It may go further and be designed to overcome some functional disability or to reestablish the patient's capacity for social or industrial usefulness. The desired end is achieved by the active participation of the patient; he has to learn to help himself. It is obvious that if occupational therapy is to be used, the needs of the patient must be understood and the therapy must be designed to satisfy them. If diversion alone is sought, many patients in any one institution may be given the same task, but otherwise the forms of therapy may be as diverse as the medicinal, physical, or other therapy. This throws an added burden on the medical attendant and he must be able to delegate the application of the therapy to a trained person. This person, or aide, should have a wide experience of human nature; he should be adaptable, patient, and prepared to take infinite pains. A much more detailed discussion might be undertaken dealing with the possible results of occupational therapy, its economic aspect and so on, but there is no need to do more than direct attention to its value. It must be emphasized, however, that to assess the value of the results of occupational therapy on a haphazard and amateurish application of its principles is as wrong as to judge any other form of therapy on the results obtained by someone who does not understand it.

From America comes an account of an attempt that is being made to carry occupational therapy to its logical conclusion. C. C. Burlingame and C. P. Wagner read a paper before the Section on Nervous and Mental Diseases at the annual meeting of the American Medical Association last June, in which they described a method of treating mental patients that was being adopted by them at the Hartford Retreat.[1] This psychiatric hospital has been treated by them as an "institute of learning". They have proceeded on the view that "fundamental progress could best be made by first regarding all psychiatric hospitals as places for education and reeducation of individual patients and that pedagogic methods under medical guidance should eventually dominate these institutions for both the acute and the chronic patients, not primarily to increase the knowledge of the patient, but for the development of personalities and the emotional stabilization through training and rational psychotherapy". They have had to adapt the whole institution to this idea; and the whole of the staff has had to be trained to accept the theory and to equip itself for carrying it out. Regular instruction is given at certain hours arranged on a time-table. Courses in business, economics, finance, insurance and government are offered; bookkeeping, secretarial work and typewriting are taught; classes in dress designing, domestic science, interior decorating, dietetics and many other subjects are held. Art classes are formed, and sketching, clay-modelling, sculpturing and painting are taught. Shops are provided so that the patients can move about and live as if they were not inmates in a hospital; even mannequin parades are staged. A library is provided and instruction in music and history is given. There seems to be scarcely an aspect of human activity that is not covered by the scheme. The authors' tables have been omitted from the article owing to lack of space (the tables will be published with the authors' reprints); no information is therefore given regarding the number of patients in the institution nor the type of mental illness from which they suffer. The lack of this information makes it impossible to offer any extensive criticism of the scheme. At first sight it would be regarded by most medical investigators as Utopian in the extreme, as a paper plan impossible of fulfilment. Most people would regard it as impracticable on the score of expense, if for no other reason; the cost of providing teachers in every possible educational subject and in every cultural field would be prohibitive. At the same time, for certain mental conditions, the underlying idea is sound. In certain circumstances we should have to agree with Burlingame and Wagner when they state:

> The patient is never completely insulated from the normal processes of living and he is encouraged to continue making his own decisions and to develop his own initiative . . . . It is a small gap to step . . . to life in the outside world.

They do not use the term occupational therapy because they think that their scheme embodies a much broader concept; they regard it as a reeducation, a training for the competition of life. It is, however, occupational therapy of the highest order. It would be of interest to have fuller information of the work carried out at Hartford Retreat, and also to hear of any work of a similar type that is being carried out elsewhere.

[1] *The Journal of the American Medical Association*, November 9, 1935.

Current Comment.

CHORDOMA.

THE notochord (*chorda dorsalis*) or primitive skeletal axis is formed from the anterior extremity of the primitive streak. As development proceeds, the notochord, as a distinct structure, is lost, but remnants persist in the pulpy centres of the intervertebral fibro-cartilages. The remnants are termed *nucleus pulposus* and are confined under tension, acting as shock absorbers for the vertebral column. The vertebral column is developed around the notochord, except at its anterior end, which is embedded in the *basis cranii*. To form the intervertebral disks the mesoblast between the vertebral bodies becomes fibro-cartilaginous. At the same time the notochord, disappearing elsewhere, here enlarges and its cells form an irregular network in the central intervertebral pulp, and here remnants are found throughout life. The notochord extends through the *basis cranii* from the anterior margin of the *foramen magnum* into the posterior part of the body of the sphenoid, immediately below the anterior end of the mid-brain. This has been termed by Gegenbauer the vertebral region, and the presence of the notochord there suggests that the region was primitively vertebral in origin. As the

notochord passes through the *basis cranii* it pierces the basilar part of the occipital bone from within outwards towards Rathke's diverticulum. Lying on the ventral surface of the occipital bone, it blends with the buccal epiblast in the dorsal wall of the pharynx. Accordingly some dorsal pharyngeal tumours may be due to proliferation of notochordal remnants. In this category may be placed craniopharyngiomata. The notochord finally pierces the basilar part of the occipital bone from without inwards, to end near the pituitary body and *dorsum sellæ*.

Chordoma is a tumour arising from notochordal remnants. Virchow, in 1856, first described such tumours, and two years later Müller elucidated their true nature. Up to the present time over 125 cases have been recorded. An interesting case and a discussion on ætiology were published by H. S. Newland and H. H. Woollard in *The Journal of the College of Surgeons of Australasia* in November, 1929.

H. S. Coulthard and R. I. Harris state that in injury to the cartilaginous intervertebral disks the notochordal remnants escape and form extrusions into the vertebral bodies.[1] X ray examination shows an area of rarefaction in the vertebral body surrounded by a denser ring. It is generally held that chordomata arise from notochordal remnants, but how this occurs has not been determined. The exciting cause is unknown, but injury, as usual, has received some blame. In the vertebral column chordomata are most frequent in the cervical region, less so in the lumbar, and rare in the dorsal vertebræ. They also occur in the sacro-coccygeal and spheno-occipital regions, the jaw, cranium and pharynx. In most cases chordomata are merely of local malignancy. Very few instances of metastasis have been reported. These have involved the lungs, liver, spleen, kidneys, heart, thyreoid and skin. Growth is generally slow and diagnosis must be based on microscopic findings. Clinical diagnosis is very difficult, except in such as involve the sacro-coccygeal area. When the vertebræ are affected, tuberculosis may be suspected, and even X ray examination may not differentiate between them. Chordomata may be mistaken for metastatic new-growths and even when involving the sacro-coccygeal area may be diagnosed as sarcomata; but usually the growth of a chordoma is far slower than that of sarcoma. Coulthard and Harris insist that chordoma should always be considered when any pelvic mass arising from the sacrum is discovered. Unfortunately surgical removal is nearly always followed by local recurrence. X ray applications may give temporary relief.

Coulthard and Harris report three cases in adults, aged thirty-six, forty-nine and fifty-three years, two being males. All were fatal. None showed metastases. In the first case the twelfth dorsal vertebra was affected and injury sustained six years previously was assigned as the cause. There was severe pain in the back, radiating to the front of the abdomen and down the thighs. Pott's disease was diagnosed, but negatived by X ray examination. A peculiar mottled appearance was shown throughout the body of the twelfth dorsal vertebra, which was compressed from above downwards. The adjacent intervertebral disks were unusually thin. Ten months later mottling was seen in adjacent parts of the eleventh dorsal and first lumbar vertebræ, the intervening disks having disappeared. Ascending motor and sensory paralysis developed with paraplegia up to the level of the umbilicus. Swellings developed to the left and right of the twelfth dorsal vertebra. A piece removed from one of these tumours showed the typical large bladder-like, physaliphorous cells of Virchow. The second case was a presacral tumour. X ray examination showed destruction of much of the anterior surface of the sacrum, with a partially calcified tumour projecting anteriorly into the pelvis. Autopsy disclosed a soft, boggy, friable tumour with zones of necrosis and many fragments of bone throughout its tissues. Typical bladder-like cells were less evident than in the previous case. In the third case there was a large tense and elastic mass on the anterior surface of the sacrum and a similar tumour under the left *gluteus maximus*—apparently an extension of the primary tumour through the great sciatic notch. X rays revealed marked destruction of the lower part of the sacrum. Much more research is necessary for the understanding of these neoplasms.

[1] *The Canadian Medical Association Journal*, November, 1935.

THE SOUTH AFRICAN JOURNAL OF MEDICAL SCIENCES.

IN spite of the enormous number of medical journals already in existence we welcome the appearance of the first issue of *The South African Journal of Medical Sciences*. This journal, published by the Witwatersrand University Medical Graduate Association, is to be devoted to purely scientific aspects of medicine. The need for a scientific medical journal exists in South Africa and recently an attempt has been made to satisfy that need by the publication of quarterly scientific numbers of *The South African Medical Journal*. We have always held that the scientific work of a country, medical or non-medical, should be published in that country; a great deal of the research carried out in Australia is issued to the world through the pages of this journal, and we hope that the new journal in South Africa will receive the support of scientific workers in that country. At the same time we should be sorry to see all purely scientific material diverted to the new special journal from *The South African Medical Journal*, which is a general practitioner's journal. General practitioners must be provided with a mixture of the scientific and the practical if they would improve their knowledge. The first number of *The South African Journal of Medical Sciences* contains articles of a high standard. We wish the new journal every possible success; its editors are Joseph Gillman, I. Liknaitzky, and Dighton Stammers.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Renal Denervation.

IN 1932 G. Milles, with two other workers, carried out experiments on dogs that had been subjected to unilateral denervation of the kidney. They showed by means of skiagrams after injection of a suspension of bismuth oxychloride into the renal vascular bed that: (i) dilatation of the renal vascular bed occurred after denervation; (ii) the vascular bed of the denervated kidney did not contract after the intraarterial injection of epinephrine; (iii) the denervated kidney, unlike the normal kidney, was not damaged by repeated chilling; (iv) a single injection of a toxic agent, such as snake venom, caused greater changes in the denervated than in the normal kidney. Together with M. Hardgrove, G. Milles has extended his previous work (*Archives of Pathology*, October, 1935). They have endeavoured to determine the response of the denervated kidney when it is exposed to various agents over long periods. Unilateral renal denervation was performed on dogs, and after a period of two weeks daily intravenous injections of colon bacilli were given to one group of animals, injections of pitressin to another group, and injections of colon and pitressin to a third group. In more than half of the animals surviving for a month fibrosis associated with chronic inflammation was found in the denervated kidney. The changes involved the interstitial tissue, the basement membrane of Bowman's capsule and particularly the arterioles. After denervation the renal vascular bed undergoes a period of dilatation and improved circulation lasting but a few weeks or months. This gives way to a period of dilatation and stasis. The authors hold that these vascular changes brought about a lowering of resistance of the renal tissues to the noxious agents used in their experiments.

Liver Changes in Exophthalmic Goitre.

G. R. CAMERON AND W. A. E. KARUNARATNE (*The Journal of Pathology and Bacteriology*, September, 1935) have endeavoured to determine whether there is a true association between exophthalmic goitre and liver damage independent of venous stasis. The material examined by them was obtained from thirty proved cases of exophthalmic goitre at University College Hospital, London. Apart from passive venous congestion the authors recognize three types of liver change associated with exophthalmic goitre: (i) cases with acute damage (marked fatty change and/or acute liver necrosis); (ii) cases with evidence of progressive damage (various stages of cirrhosis); (iii) arrested cases (nodule formation, atrophy). They regard these changes as representing reactions to varying grades of injury the result of the thyreoid derangement. They point out that several observers have attempted to connect the toxic changes found in the liver in exophthalmic goitre with a disturbance of the glycogenic function. The authors have tested this hypothesis by certain experiments on albino rats and could find no evidence to support it.

Liver Regeneration and Biliary Obstruction.

ON the basis of his experience that autoplastic grafts are very sensitive indicators of the growth capabilities of liver and bile duct cells as they exist *in situ* at the time of transplantation, G. R. Cameron (*The Journal of Pathology and Bacteriology*, September, 1935) has carried out experiments to determine whether, after biliary obstruction has been produced, liver cells can still grow when transplanted into a suitable nidus, such as the animal's own omentum. One hundred albino rats were used in the investigation, and the author describes the technique used by him. Grafts were made after obstruction of the common bile duct and after obstruction of the duct when the obstruction had been relieved. The author found that the liver cells were so affected by the obstruction that they were unable to grow when transplanted; bile ducts were unaffected. He points out that his study confirms the impression that biliary obstruction results in interference with the regenerative capacity of the liver cells, even at a very early period after the onset of obstruction. The disturbance is for some time temporary, being followed quickly by a return of the normal capacity to proliferate. Since bile ducts suffer no alteration, surviving and growing in grafts in much the same fashion as under normal conditions, fresh evidence is brought forward of the relative independence of liver cells and bile ducts.

Hæmangioma of Skeletal Muscle.

ROBERT MAILER (*The British Journal of Surgery*, October, 1935) reports a case of hæmangioma of the triceps occurring in a man, aged twenty-one years. The tumour was non-encapsulated and consisted of dilated capillaries and thick-walled arterioles in a young fibrous connective tissue stroma. The author points out that the history in his case was strongly suggestive of a traumatic origin, and he believes that the pathological findings pointed in the same direction. He surveys 256 cases reported in the literature; in 43, or 17%, of these trauma was mentioned as a possible factor. The author points out that the early age incidence of these tumours has inclined most authors to regard the congenital factor as of supreme importance, with trauma as an occasional secondary exciting agent. The author has offered an explanation as to the way in which trauma acts in the production of these tumours.

E. N. MACDERMOTT (*ibidem*) reports two cases of hæmangioma of voluntary muscle, occurring respectively in a woman twenty-four years of age and a boy twelve years of age. He states that 260 cases have been reported in the literature. He holds that the most noteworthy points in the pathology of these tumours are: (i) that, while clinically benign, metastases never occurring and recurrences being very rare, they may exhibit a peculiar local malignancy, infiltrating the muscle, which degenerates before them; (ii) a tendency towards partial replacement of the originally angiomatous structure by fibrous or fatty tissue in the centre. The recurrence rate in the literature works out at 6%, but the author expresses the view that this is usually, if not always, explicable by inadequate excision.

Paget's Disease of the Nipple.

ROBERT MUIR (*The British Journal of Surgery*, April, 1935) believes that the majority of cases of Paget's disease result from an extension of an intraduct carcinoma of the surface epidermis of the nipple. In his original paper Paget noted the association of malignant growth in some other part of the breast with this condition. The author applies the term Paget's disease to a characteristic "nipple lesion liable to be associated with mammary carcinoma". He then brings forward the results of observations to show that both conditions may be secondary to the proliferation of malignant cells within the ducts of the gland. A Paget cell is defined as "a cancer cell growing within a healthy, or at least non-neoplastic epithelium". All transition stages from simple papillomatous ingrowths in the ducts to intraduct carcinoma are found. Ultimately the duct may be filled with a mass of cells resembling encephaloid carcinoma. These cells may completely fill the duct, extending even into the acini. This intraduct carcinoma is of frequent occurrence and may occur in a localized area in association with local induration or cyst formation. The disease occurs at a younger age than does ordinary carcinoma. It may be present for many years without giving rise to infiltrating carcinoma. The author believes that the Paget cells extend towards the surface of the nipple by multiplication between the layers of relatively healthy epithelium lining the ducts; this he terms "the intra-epithelial spread of carcinoma". Similar intra-epithelial spread has been recorded by other workers in describing malignant melanoma. On reaching the surface of the nipple the Paget cells undergo transition to a columnar type and continue to extend beneath the surface

epithelium, thus producing the characteristic eczematous condition. If intraduct carcinoma invades the breast stroma, the ordinary infiltrating carcinoma is produced.

Papillomatosis Peritonei.

ARTHUR H. WELLS (*The American Journal of Pathology*, November, 1935) reports the discovery of a widespread villous type of benign peritoneal papilloma at the autopsy of an elderly man who died four hours after the first operation for prostatectomy. The papillomata were scattered over the parietal peritoneal surfaces, being concentrated chiefly on the diaphragm, omentum and mesentery of the small intestine. They varied in size from slight irregularities, invisible to the naked eye, to lesions nearly a centimetre in diameter, and they occurred singly or in small groups. The opened intestines contained no sign of neoplasm and no single primary source of the peritoneal lesions could be found.

MORPHOLOGY.

The Ultimobranchial Body.

B. F. KINGSBURY (*Anatomical Record*, January, 1935) states that a detailed examination of forty selected human embryos, 13 to 55 millimetres in length, has furnished him with no conclusive evidence of the development of thyreoid parenchyma from material contributed by the caudal pharyngeal complex. On the contrary, the evidence has indicated that after fusion with the median thyreoid gland the complex undergoes reticulation and progressive degeneration. Thus there seems to be no justification for substituting the term "lateral thyreoid" for the more usually employed "ultimobranchial body". Certain general and comparative considerations are discussed, considerations related to the fate of the ultimobranchial body within the thyreoid.

The Innervation of the Kidney, Ureter, Testicle and Epididymis.

G. A. G. MITCHELL (*Journal of Anatomy*, October, 1935) holds that many failures following operation on the autonomic nervous system have been due to inaccurate anatomical knowledge. He gives a description of the autonomic nerves and plexuses from which the nerves to the kidney, ureter, testicle and epididymis are derived. His article is illustrated by a series of diagrams and photographic plates. The kidney receives a nerve supply from: (*a*) the aortico-renal ganglion, (*b*) the middle and inferior splanchnic nerves, (*c*) the intermesenteric nerves, (*d*) the lumbar sympathetic chain. Stripping of the renal artery will not secure complete renal denervation in every case. The ureter receives filaments from: (*a*) the lower fibres of the renal plexus or the intermesenteric nerves, (*b*) the superior hypogastric plexus or the upper end of the hypogastric nerve, (*c*) the lower end of the hypogastric nerve and the upper part of the inferior hypogastric plexus. The inferior ureteric nerves are closely associated with filaments which supply the *vas deferens* and seminal vesicle. The testicle and epididymis receive nerves from: (*a*) the intermesenteric nerves and renal plexus, (*b*) the superior hypogastric plexus or the upper end of the hypogastric nerve, (*c*) the inferior hypogastric plexus. Testicular afferents apparently enter the cord at a higher level than is commonly stated. In one dissection the author discovered a direct communication between the superior testicular nerve and a nerve supplying the third part of the duodenum. He thinks that such a nerve may explain the sickness associated with testicular injuries. It is probable that the nerve supplies to the testicle and epididymis are distinct and separate.

The Formation of the Venous Valves, the Foramen Secundum and the Septum Secundum in the Human Heart.

P. N. B. ODGERS (*Journal of Anatomy*, July, 1935) has studied in human embryos the formation of the venous valves, the *foramen secundum* and the *septum secundum* in the human heart. He concludes that while the right venous valve is formed by the coaptation of the wall of the right horn of the *sinus venosus* with that of the auricle, and grows by the increasing invagination of the former into the latter cavity, the left valve has its origin in a delamination of the *septum primum*. The appearance of the *foramen secundum* is due to a local failure of growth of the *septum primum*, rather than to a breakdown of the septum already formed. The *septum secundum* has a double origin. Not only is there a dorsal outgrowth in association with the upper end of the left venous valve, but there is also a ventral proliferation from the *septum intermedium*, commencing to the right of the anterior inferior angle of the *septum primum* and blending later with the sinus septum and the lower end of the left venous valve.

The Anatomy of the Major Duodenal Papilla.

VINCENT J. DARDINSKI (*Journal of Anatomy*, July, 1935) has investigated the musculature of the major duodenal papilla of man. He bases his conclusions on the study of the duodenal papillæ and associated ducts in one hundred cases. He concludes that the greatest constriction of the common duct is produced by the intestinal muscle. This would, in the author's opinion, seem to be the most logical point of the sphincteric action, if there is any, and the constriction would seem to be produced by the intestinal muscle. The independent muscle fibres, described by Oddi as being responsible for sphincteric action, are not circular in their arrangement, but oblique, and the point of greatest constriction of the common duct is not outlined by these fibres. The longitudinal mucous folds within the papilla have a definite arrangement, and the presence of muscle fibres in these folds indicates that they have an active part. The mucous glands present are direct outgrowths of the epithelial lining of the papilla. The author offers a hypothetical, but, as he states, a logical, explanation of the function of the duodenal papilla; his explanation is based on the structure of the papilla and its muscular arrangement.

Omental Lymphatics in Man.

PARKE H. SIMER (*The Anatomical Record*, October 25, 1935) has investigated by india ink injections the distribution and connexions of the omental lymph channels of man. He finds that lymph channels accompany the blood vessels in all parts of the omentum, but that they are in most preparations hidden by the perivascular fat. India ink injections reveal a dense plexus surrounding the artery. Lymph passes into longitudinal collecting vessels which lie near margins of the fat strips. Almost all the lymph from the omentum is carried by collecting vessels into the gastro-epiploic lymph glands. The splenic glands are of minor importance. Lymph from the primary collecting centres passes to the cœliac glands and then into the *cisterna chyli*.

The Nerve Cells in Spinal Ganglia.

D. M. BLAIR, P. BACSICH AND F. DAVIES (*Journal of Anatomy*, October, 1935) have examined the nerve cells of the spinal ganglia of the cat by many histological methods, including the prolonged osmic acid method, toluidin blue and various silver techniques. They differentiated three types of ganglion cells—large round pale, small round pale, and dark multangular cells. By microincineration and the study of the Golgi apparatus they demonstrated the separate morphological identity of the dark cells. These dark cells differ from those of the sympathetic ganglia in being rarely multipolar. The authors bring forward evidence to show that the dark cells are not the product of shrinkage or degeneration. The different appearance of the dark cells from that of the pale cells is presumably due to a different physicochemical constitution, which may be associated with a different function. The authors point out that certain internal structural features of the dark cells of spinal ganglia are among those recognized by other workers as typical of motor nerve cells; on this point, however, they reserve judgement pending the outcome of further experimental studies. The authors' paper is illustrated by a series of excellently reproduced photomicrographs.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on October 31, 1935, DR. A. M. DAVIDSON, the President, in the chair.

Cerebral Trauma.

DR. C. G. MCDONALD read a paper entitled: "The Sequelæ of Head Injuries" (see page 45).

DR. GILBERT PHILLIPS read a paper entitled: "The Surgical Sequelæ of Acute Cerebral Trauma" (see page 41).

DR. A. M. DAVIDSON, in declaring the subject open for discussion, said that the responsibility of surgeons in the present day had been increased by the dangers of traffic and that many young people were the victims of head injuries. The appallingly high incidence of such injuries made the subject one of the utmost importance.

DR. OLIVER LATHAM said that he had been asked to say a few words on the pathology of head injuries and their sequelæ. He confessed that his own ideas had not always been too clear, and it seemed that there were others so situated. Thus Wertheim wrote: "In the mode of development of these lesions—their relations to the trauma and to the clinical picture, and indeed in the whole question of trauma and residual states—there reigns the greatest imaginable confusion." Not concerning themselves with gross smashing causing obvious direct brain damage, they would busy themselves with less obvious lesions. They had long recognized brain bruising directly beneath the part struck—swelling and discoloration of the brain tissue *plus* small hæmorrhages in the brain substance. They had also recognized the effects of *contre-coup*, with its different picture of pial vessels torn and subpial hæmorrhage. This hæmorrhage might continue and thus cause a cerebral hæmorrhage out of all proportion to the original trauma and lesion. Contusion therefore was a local state, concussion a remote phenomenon.

The real trouble was to explain in pathological terms late hæmorrhages weeks and months after the trauma, as well as papilloedema, naked eye and microscopic softenings and demyelinations, the increased intracranial tension which could lead to cranial nerve dysfunction, and clinical and mental phenomena with no apparent lesion at all. Ritchie Russell pointed out that there was always cerebral œdema, and Trotter that this, unresolved, caused most of the concussion symptoms. They had to admit, however, that cerebral œdema was common, that fresh perivascular and capillary hæmorrhages and their residuals (*"états vermoulus"*) might all be present without giving rise to any clinical signs. With Russell they had to believe that in severe concussion the whole brain suffered, and Russell argued thus: "We know that animal brains punctured with sharp instruments heal with least neuronal damage and glial or scar formation and clinical trouble. Indeed warriors have fought on with thin blades in their brains. It is quite a different story with blunt instruments, where great glial scar tissue with neuronal and functional phenomena obtains. On the other hand, we can have traumatic unconsciousness without a visible lesion."

Russell suggested the following mechanism. The brain tended to be trajected as a heavy mass and rebounded in the opposite direction, the latter movement being at times the worse. A force on the outer diploe became at the inner diploe greatly diffused. Surface tension and higher intracranial pressure kept the dura closely applied to the skull, and the pia-arachnoid to the dura. However, the brain could get further away from the arachnoid, since fluid can nearly always be found to fill up vacuums there; hence pial bleedings. This diffusion of force was shown by widespread capillary hæmorrhages after a definite circumscribed tap. Falls in special directions would bring the mid-brain up against the *falx cerebelli*; hence bruising, jarring, hæmorrhage and softening occurred here. So much for this aspect. The increased intracranial tension and brain swelling evoked many theories. All authorities seemed inclined to name the vasomotor centre in the medulla as the culprit. Differences in the hydrogen ion concentration or the jarring were said to upset its functioning. Thus Wertheim held that there was a growing recognition of the significance of functional disorder of the blood vessels, and this was giving a new angle to the conception of the condition and thus changing the foundations of a large part of the neuro-pathology of concussion. And again, Wertheim held that vascular lesions played a large part, but there was no specificity here; similar pictures obtained in other diseases. Hassin held that vasomotor alterations caused vascular retardation and suspension of exchange between blood and tissue fluid, and thus necrosis—a physiological theory as against an anatomical one of parenchyma affected. Weil held that in the disturbance of the vasomotor mechanism or regulation they might find an explanation of the sudden hæmorrhages into the brain weeks, it might be months, following a concussion with scarcely a symptom. Weil also suggested that sudden extension of vasomotor nerves (especially at the point of entrance of cerebral and spinal vessels into the skull or vertebral canal) caused irreparable damage to the vessel walls—vaso-dilatation, stasis and focal anæmias.

In regard to jarring, Jacob and others held that mere jarring could destroy or damage neurones singly or in batches. Dr. Latham was going to ask those present to transfer their interest to the spinal cord, the finite bounds of which allowed more exact and comprehensive study. Gordon Holmes, among others, called attention to that interesting axonal degeneration (compression myelitis) of the anterior horn cells *et cetera* which might occur in trifling wounds of the vertebra, in cancers, tuberculosis and other pathological states near, but not involving, the spinal canal—at least to the naked eye. But they had to postulate some alteration in the exchange of tissue fluids there, because there could have been no question of jarring in a boil-affected patient whose cord, showing this condition, Dr. Inglis had sent to Dr. Latham. This axonal degeneration included eccentric nucleus, loss of tigloid bodies, "myelitis" and swelling of the axis cylinder, and ultimately disappearance of the neurone. Gordon Holmes found this neuronal change also in the occipital cortex after slight bullet wounds leaving no external trace, and it had been found near and away from those capillary hæmorrhages in trauma and apart altogether from hæmorrhages; there was surely enough to account for concussion states.

What caused these neuronal obituaries? Quite a valuable book had been written describing the myelitis which could be revealed by Marchi's staining caused by roughly handling a spinal cord on removing it from the body. Granted that the ischæmia caused by an armlet might cause functional disturbance in a nerve, this soon recovered. Conversely, the time taken by the nerve to recover (if it recovered at all) after handling and compression depended on the extent and duration of the insult or tap.

Some further pictures might be obtained by examining the tracks of bullet wounds in the brain. The first zone showed cerebral tissue, non-staining and œdematous. The next zone showed hæmorrhage and damaged brain, while the most distant or remote showed multiple small hæmorrhages round the capillaries, and ischæmic changes in the ganglion cells. As a rule, glial proliferation was a later reaction. Similar conditions were found in experimental monkeys.

Meyer described multiple microscopic areas of softening in cases of traumatic insanity, and here it was necessary to allude to Martland's clinical condition, "punch drunk", wherein the boxer developed slowness of movement, speech defects, hand tremors, leg dragging, staggering and Parkinsonism, evidently with an organic basis like Meyer's (above). Again must be stressed neuronal death apart from hæmorrhage, small or great. Penfield stressed widespread glial reaction in these late lesions. It seemed right here to refer to Bruetsch and De Almond's case of Parkin-

sonism occurring after a fall; the *post mortem* examination two and a half years later showed demyelination, "*état vermoulu*", and obvious neuronal loss in mid-brain and brain stem and cortico-spinal tracts, definite leg paralysis being present immediately after the fall; yet the man returned to work. They had to remember that age influenced the picture. Prognosis was bad after forty, while gross brain deformities in children might resemble primary or intrinsic developmental disorders, the reparative histology being so good in the young.

DR. S. SHELDON said that he had been disappointed in the paper by Dr. Phillips in that no indications had been given as to when operation should be performed. He had had some considerable experience of injury to the brain. Since February, 1928, he had performed 400 *post mortem* examinations for fractured skulls, not including injury to the brain without fracture, which was common. He asked members to consider what happened when fracture of the skull occurred. In his experience, fracture of the skull was unimportant *per se*, unless it was a depressed fracture. If a person got a bump on the back of the head the dura became separated, usually by extravasation of serum, and would strip off. But the injury that appealed to him as being of importance was that occurring by *contre-coup*. Cases of so-called concussion in his experience always showed damage to the brain, usually in the anterior fossa. The brain was contused and, even months or years after, was seen to be scarred and softened. The only cases that would be benefited by operation were those in which there was middle meningeal hæmorrhage. But it was difficult to say whether the bleeding was on one side or the other. If the patient got a smack that would break the temporal bone, he often got an injury on the other side of the head.

Dr. Sheldon did not recognize the subdural hæmorrhages of which Dr. Phillips spoke. He could not say that he saw them localized as Dr. Phillips had shown in his diagrams. The consequence of subdural hæmorrhage was considerable bleeding, which extended to the base, and clotting took place between the dura and the arachnoid. *Pachymeningitis hæmorrhagica interna* was no doubt a very infrequent consequence in those cases which came to *post mortem* examination. But it might often occur in patients who recovered. There was no doubt about the organization that took place. Membranes or curtains were formed between the dura and arachnoid.

Dr. Sheldon commended the judicious way in which Dr. McDonald had dealt with the question as to whether symptoms were neurotic or organic after injuries. From experience he was of the opinion that the symptoms complained of were nearly always genuine. Agrophobia and so on might be functional, but Dr. Sheldon thought that they were grounded in an organic lesion. In conclusion Dr. Sheldon said that on several occasions he had seen intrapontine hæmorrhage as a result of injury to the brain.

DR. LYLE BUCHANAN said that the lectures were illuminating and had given food for thought. The pathology of subdural hæmatomata was subject to much controversy. Dr. Buchanan urged upon surgeons and physicians the wider use of the technically simple procedure of encephalography. In many cases of cerebral trauma the patient's condition was improved by medical treatment and the use of encephalography. Headaches diminished and the mental outlook improved. Dr. Buchanan said that patients even came back to him after a year had elapsed and asked him to perform encephalography again. So far, in a series of over forty encephalographies done for this condition, there had been no resultant mortality or morbidity, and many patients showed definite clinical improvement. The technique and apparatus were simple. All that was required was a ten cubic centimetre syringe, a three-way tap and a manometer attached to a three-way tap. The patient should spend three or five days in bed. Encephalography was a valuable aid in diagnosis; it proved or disproved the presence of hæmatoma. Dr. Buchanan insisted that the remarks about the lack of danger did not apply to encephalography done for cerebral tumour.

DR. DOUGLAS MILLER remarked that Dr. Phillips had spoken of massive acute subdural hæmatomata which occurred as a result of accident; but he (Dr. Miller) questioned the classification of this condition as a surgical sequela, namely, one in which the surgeon could do something to improve it. He quoted Jefferson, who stated that the primary mortality from such conditions appeared to be unavoidable. In a series of ninety deaths, 60% of patients died in the first twenty-four hours. A large number of these patients had massive subdural hæmatoma; but the concomitant damage was such as to render operation of no use. It appeared that operation was only likely to be useful for hæmatoma when the patient survived the first twenty-four hours. The hæmatoma in the patient who survived twenty-four hours was usually becoming encapsulated and formed a single problem.

Dr. Phillips had said that there was some confusion as to the classification of subdural hæmatomata. If there was confusion, it was shared by Trotter and all other eminent surgeons who had written on the subject; Dr. Miller was not aware that they had drawn attention to the various classifications made by Dr. Phillips. Dr. Miller also mentioned the experiment, described by Dr. Phillips, in which the intact capsule of the subdural hæmatoma had been bathed in fluids of varying density and from which the conclusion had been drawn that the membrane was permeable and able to absorb cerebro-spinal fluid. If, as Dr. Phillips described, some of these lesions were extra-arachnoid and subdural, they would not be bathed by cerebro-spinal fluid at all. The attempt to divide lesions into extra-arachnoid and subarachnoid was, he thought, rather inviting confusion than clarifying.

DR. H. S. STACY wished to join issue with Dr. McDonald as regards classification. He followed the classification of Trotter and others, who stated that any patient with concussion was well in twenty-four hours. To speak of fatal cases of concussion was to speak at variance with the views of leading British surgeons. Patients with head injury were admitted to hospital under the care of surgeons; the surgeon saw them in the beginning, but frequently not in the end, and the surgeon would in some cases come to a conclusion different from the physician's or the medical board's. Dr. Stacy agreed with Dr. Sheldon that a minority of cases were functional. He described the typical picture of a man with head injury who had very little to show and few focal signs, but who was altered in manner and had insomnia, loss of memory, perhaps homicidal tendencies and other vague symptoms. The easy way was to say that his condition was functional; there was no question of that. Dr. Stacy thought that lesions were too often classed as functional when further investigation would show them to be organic.

Dr. Phillips had drawn attention to Jefferson's statement that epilepsy was ten times more common when the dura was penetrated. This might explain J. B. Murphy's opinion that a frequent cause of Jacksonian epilepsy was adhesions between the dura and arachnoid. Dr. Stacy said that he had separated adhesions and interposed a piece of dura, as suggested by Murphy; he had had some good results and some bad.

Dr. Stacy said that subdural hæmatoma was a fascinating question. He spoke of a patient who, some time after a head injury, came back suffering from a carbuncle of the neck and weakness of one leg. A physician expressed the opinion that the paresis was due to spinal myelitis, the result of infection from the carbuncle. Careful inquiry elicited the fact that the patient had never been well since the original head injury. The patient died a few days later and *post mortem* examination revealed a subdural hæmatoma. This case was taken to law and with some difficulty Dr. Stacy was able to show that death was due to the original injury.

DR. W. S. DAWSON thought that a great deal of the confusion regarding organic and functional arose through failure to give full value to the symptoms manifested. Headache and other symptoms should not be taken at their face value without investigation. Nobody would deny that true loss of memory was due to organic injury. On the other hand, in neurotic patients headaches might

be diffuse and merely cause discomfort; they were associated with anxiety and were different from the type of headache due to an organic lesion. Symptoms should be looked at more critically. Dr. McDonald pointed out the importance of attending to symptoms at the earliest possible moment. The surgeon might hand the patient over to his medical colleague when the injury had healed, and the physician, by suitable treatment, might prevent a neurosis.

Dr. Dawson asked Dr. Phillips about the epileptic sequelæ of head injuries. In reference to the case mentioned, in which a cyst had been discovered some years after the original trauma, he wondered whether, after the operation had been performed, there might not still be risk of glial formation after a further latent interval, with recurrence of fits, although the immediate result had been satisfactory.

DR. R. A. MONEY referred to the pathology and terminology of intracranial hæmatomata. All were agreed on the suitability of the old terms, and he did not see the force of Dr. Phillips's idea in introducing new terms. Dr. Money thought that it would be clearer to stick to the old terminology; they then knew what they were talking about.

In regard to diagnosis, all lesions should be regarded as organic until they could be proved to be functional. Dr. Money said that if the headache were aggravated by exertion, an organic cause was suggested. Another guiding symptom was the variability of the headache. The neurotic patient would say that it was always there; when the pain was real the patient admitted to variability. This was especially so in the subdural type of hæmorrhage.

In estimating the pressure of the cerebro-spinal fluid Dr. Money placed reliance on a simple manometer, which, he thought, should always be used when lumbar puncture was performed. By this means it was possible to obtain valuable information and records on which reliance could be placed. He had been surprised at the inaccuracy of the ordinary methods of estimating intracranial pressure. Another aid to diagnosis was encephalography. But he thought this was a serious procedure, though safe if performed by skilled hands and by a standardized method. The patient should be in an upright posture in a chair with an adjustable headpiece. Dr. Money advocated a method of continuous flow and replacement, rather than the use of a ten cubic centimetre syringe. There was no need to take the pressure, because a little fluid ran in and an accurate amount of air could be put in and directed to where it was needed. Encephalography was of value more in its negative than in its positive findings. It was possible by these means to reassure the patient. Dr. Money spoke of the sequelæ brought about by the patient's having gaps in the bony cranium. Such a person was an object of curiosity and was constantly warned of the dire results of possible injury. Therefore he became an invalid before he started to do anything at all. Dr. Money thought that the gap should be filled in, not by silver or celluloid, which would make him even a greater object of wonder, but by bone. The patient would then be regarded as normal. For grafting Dr. Money recommended the use of the inner table on the ilium. He had this idea from Pickerill's article which appeared in THE MEDICAL JOURNAL OF AUSTRALIA. Dr. Money had operated on five such patients successfully; they had all become normal.

Dr. McDonald said, in his reply, that he disagreed with Dr. Buchanan's statement that the victims of head injury might be treated efficaciously with encephalography. All members present would judge for themselves whether this was advisable therapy, but Dr. McDonald deprecated its use, particularly in cases of traumatic neurosis. The less surgical interference in these cases, the better for the mental health of the patient. Dr. Stacy had disagreed with his assumption that the symptoms of many patients who had sustained concussion were manifestations of a neurosis; and he was in good company, notably that of Dr. Trotter, who was a great authority and a distinguished surgeon. Nevertheless the dicta of the British neurological surgeons, which Dr. Stacy had described as authoritative, were subject to criticism, like all other opinions in medicine. The physician examined almost as many of these patients as the surgeon, but he saw them at a later stage, and he had the means which the surgeon had not, or did not choose to have, of inquiring patiently into the symptoms, of talking with the patients and, in short, of taking a complete history. This taught a great deal. Dr. Stacy had said that Dr. Sheldon had made the statement that the majority of these patients were not neurotic. He had not heard Dr. Sheldon say that. What he did hear Dr. Sheldon say was that almost all these patients were honest. On that point Dr. Sheldon and he were in complete accord. Dr. Stacy had contended that a patient manifesting loss of memory, homicidal tendencies and other serious symptoms must have an organic lesion. With this all would agree, and it would be foolish to deny it. But the occasional occurrence of grave mental changes after injuries to the head was no justification for the assumption that every patient complaining of dizziness and pains in the head must have a persistent contusion of his brain.

Dr. McDonald supported the excellent advice given by Dr. W. S. Dawson. By a careful study of symptoms just as much could be learned of the manifestations of disease as by frequent visits to the *post mortem* room. Finally, he congratulated Dr. Money on his remarks, which showed that Dr. Money, although a surgeon, appreciated fully the psychological factors associated with head injury. He was also glad to hear him say that the taking of an encephalogram was a serious procedure. Like Dr. Money, he believed that a diagnosis of traumatic neurosis should be made only when the evidence was strongly against the existence of an organic lesion, but this did not mean that the diagnosis should be made rarely.

Dr. Phillips, in reply, said that he advocated sweet moderation. Dr. Sheldon asked when should operation be performed? It was difficult for him to reply in the presence of Dr. Storey, who was said to have tattooed across his back the words: "Please do not lumbar puncture". Dr. Phillips agreed that lumbar puncture was a serious procedure; but it had proved in skilled hands to be a safe procedure.

Dr. Phillips disagreed that the volume of air injected into the lumbar sac should be rigidly determined and a certain amount given in a certain time. Lumbar insufflation of air sometimes benefited the patient; but for a therapeutic effect a considerable quantity had to be injected and a moderately severe reaction was to be expected. On the other hand, the diagnostic value of the procedure did not depend on the injection of a large quantity. There was never need for injecting more than ten cubic centimetres of air to display the outline of the two lateral ventricles in a skiagram. It was possible by this means to examine the position of the *septum pellucidum* and the outline of the anterior horns of the lateral ventricles. As to the value of operations undertaken, Dr. Phillips was not in a position to discuss this. He himself had excised only one cerebral scar; he had seen five such operations done, with one excellent result and two that were quite good; the other patients died. Dr. Phillips agreed with Dr. Money that the condition should be assumed to be organic until it was proved to be functional. When the physician was incapable of saying that the case was not organic, the surgeon should be allowed to inject air to see if there was any displacement of the ventricular system.

A MEETING of the Victorian Branch of the British Medical Association was held at the Alfred Hospital, Melbourne, on July 17, 1935. The meeting took the form of a series of demonstrations by members of the honorary staff of the hospital and the staff of the Baker Institute. Part of this report appeared in the issue of December 14, 1935.

Biliary Calculi and Duodenal Ulcer.

DR. FAY MACLURE showed a male patient, A.C., aged seventy-six years, who, over a period of forty years, had suffered from a feeling of fullness and discomfort in the upper part of the abdomen, with a good deal of flatulence.

but no vomiting. He had had frequent attacks of epigastric pain, sudden in onset, varying in duration from ten minutes to several hours, but he had not been jaundiced at any time. Over the last five or six years he had also suffered from periodic attacks of pain coming on one or two hours after meals; this pain was relieved by food, and when it awakened him in the early hours, as it did frequently, he could get relief by taking food. One month before his admission to hospital the pain became continuous in the epigastrium and in the back, and though nauseated and anorexic, he had not vomited, but had lost 12·6 kilograms (two stone) in weight. One week before admission severe melena had occurred. At operation the gall-bladder contained stones, and a duodenal ulcer was present on the posterior wall of the first part of the duodenum. Anterior gastro-jejunostomy, jejuno-jejunostomy and cholecystostomy had been performed.

Dr. Maclure showed another male patient, T.D., aged fifty-one years, who, over a period of twenty-five years, had suffered from recurrent attacks of pain in the epigastrium one or two hours after food, and relieved by food. In addition, he had had severe attacks of pain in the right hypochondrium, passing through to the back. The pain was described as agonizing in character, but he had not been jaundiced. Of recent years the attacks of pain became more frequent, but the main complaint had been projectile vomiting. He vomited huge quantities, as much as a gallon at a time, and recognized things in the vomitus which he had eaten on the previous day. At operation it was found that he had a perforating duodenal ulcer walled off by the gall-bladder which contained stones. The duodenum was stenosed and the stomach was dilated. Anterior gastro-jejunostomy, jejuno-jejunostomy and cholecystectomy were the procedures that had been carried out at the operation.

Congenital Stenosis of the Ampulla of Vater.

Dr. Maclure showed a girl, N.G., aged fifteen years, who had jaundice for six months after birth, which cleared up, however. During childhood she had intermittent attacks of epigastric pain, colicky in nature, associated sometimes with jaundice, clay-coloured stools and bile-stained urine. At operation the gall-blader was small, with thickened walls, and the common bile duct was grossly dilated; the ampulla of Vater was stenosed and thickened. Cholecystduodenostomy was performed, and in three months after the operation the patient had gained over two stone in weight, had been free from pain, and was able to eat anything.

Pulsion Diverticulum.

Dr. Maclure's next patient was a male, G.H., aged sixty-eight years, who for eight or nine years had been in the habit of regurgitating his food, and latterly the condition had become much worse. At operation a pharyngeal pouch had been dissected, which was a huge pulsion diverticulum extending into the thorax.

Congenital Branchial Fistula.

Another patient shown by Dr. Maclure was a girl, aged nineteen years, who had had a congenital fitsula in the right sterno-clavicular region, which discharged periodically and then healed. At times the patient noticed a peculiar taste in her mouth and discovered that, by pressure on the swelling above the clavicle, the contents were expressed into her mouth.

Congenital Facial Deformity.

Dr. Maclure also showed a girl, J.S., aged twelve years, who was disfigured by the presence of a deforming enlargement of soft tissue on the right side of the face, which Dr. Maclure thought might be due to failure of development of the mesodermal tissues. At operation portion of the upper lid had been excised and portion of the tissue of the right cheek was removed. The pathologist had examined the biopsy material, which he described as a peculiar diffuse proliferation of soft areolar and fibrous tissues, often in whorls; there was no evidence of any inflammatory process or of angiomatous changes; nerve bundles appeared to be excessive, both in number and in bulk, perhaps because of an increase in the sheath elements.

Tendon Transplant for Radial Nerve Paralysis.

DR. C. J. O. BROWN presented a patient, K.B., a male, aged seventeen years, who, on December 15, 1933, had caught his arm in a machine and fractured the radius. A block of muscle was torn away from the upper part of the extensor supinator group of muscles of the forearm and the dorsal interosseous nerve was destroyed. The treatment had consisted in immediate excision of the wound and suture; healing was by first intention and the fracture united well. Extensor paralysis with wrist-drop resulted. On July 31, 1934, tendon transplantation was carried out with the object of restoration of function. The *pronator teres* was transplanted into the *extensor carpi radialis longior* and *brevior*; the *flexor carpi radialis* was transplanted into the extensors of the thumb and index finger, and the *flexi carpi ulnaris* was transplanted into the extensor tendons of the three ulnar fingers. The transplants were all made under tension, with the wrist in full dorsiflexion and pronation, and the forearm was fixed in this position for four weeks. Reeducation was commenced at that stage, but the splint had been retained for six weeks. On May 16, 1935, the patient wrote to Dr. Brown and stated that the hand did not hinder him at all.

Fracture Dislocation of the Elbow Joint.

Another patient shown by Dr. Brown was H.T., a male, aged nineteen years. He had been admitted to hospital on March 14, 1935, following an accident in which the right elbow was severely injured when the arm was caught in a machine. The skiagram showed a posterior dislocation of the radius and ulna, with a separation of the lateral condyle of the humerus. Reduction was easily performed, but as it could not be maintained, on March 19, under local anæsthesia, the condyle had been fixed in position with a screw and the limb was put up in plaster.

Carcinoma of the Descending Colon.

Dr. Brown also showed a female patient, aged thirty-five years, who had attended first at the hospital on May 20, 1932, because of frequency of micturition and constipation for three weeks. In the following December frequency was still very troublesome and she complained of pain when the bladder was full. At that time the bowel emptied regularly. The urine was turbid with motile bacilli. By means of renal lavage with acriflavine the frequency was controlled and for the next year she attended the out-patient clinic repeatedly with minor complaints; constipation was mentioned frequently in the notes. On January 2, 1934, she was admitted to hospital with acute intestinal obstruction of five days' duration. At operation a carcinoma of the upper part of the descending colon, just below the splenic flexure, was found; a transverse colostomy was done and a month was allowed to elapse before further operation. Resection and anastomosis were then performed, but she did not stand the operation well, and on this account, after the splenic flexure and the descending colon were slipped free, the growth was anterioralized after Mikulicz's method and the patient was returned to bed. Later, the loop containing the growth was cut away and the spur of the two colostomies was crushed; finally the openings were freed and oversewn. The patient was very well when Dr. Brown saw her on July 2, 1935, though occasionally she was constipated and had urinary frequency. No abnormality of the colon was revealed by a barium enema administered at that time.

Carcinoma of the Ileum.

Dr. Brown also showed a female patient, aged forty-two years, who attended the medical out-patient department first on July 21, 1933, when she had complained of pain in the lower part of the abdomen for three or four months, coming on in attacks which lasted about four hours. Sometimes she had vomited during the attacks, but vomiting had not relieved the pain, and taking food had made the

pain worse. She was in the habit of taking medicine to avoid constipation and had some frequency of micturition during the attacks. The urine was dark brown and numerous motile bacilli and pus cells were present. The urine also contained 2% of sugar and numerous yeast cells.

On August 11, 1933, it was noted that there was some thrombosis in varicose veins of the left leg. On September 26, 1933, she was referred to the surgical clinic for cystoscopy. The urine smelt strongly of fæces and contained fæcal flakes. Cystoscopy showed that the bladder was very inflamed and œdematous and contained fæces and air; a ragged indurated area was seen near the fundus and was regarded as the site of entry of a fistula. A barium enema was given and the colon was reported to be normal in appearance.

On October 24, 1933, the patient had an operation. A carcinoma of the ileum, about 30 centimetres (twelve inches) above the ileo-caecal valve, was found; it had perforated into the bladder, to which it was adherent with several indurated adjacent loops. The ileum was divided above the growth, implanted into the side of the caecum and divided again close to the caecum, with closure of the distal end. The intervening foot of the ileum, which contained the growth and the adherent loops, was removed with the fundus of the bladder, and the bladder was sutured and drained through a catheter.

After an uneventful convalescence and healing of the wound a course of deep X ray therapy was given to the region, and on June 20, 1935, the patient reported in good health.

Dermatological Conditions.

DR. S. W. SHIELDS showed a number of patients with dermatological conditions of interest, among whom were the following.

A female patient, aged thirty-one years, was suffering from tertiary syphilis and *dermatitis artefacta*. She was undergoing treatment for syphilitic ulceration of the leg, which was responding satisfactorily. She also had a large ulcerated area on the back of the right hand, which, she stated, had developed overnight. It was obviously an artefact due to the action of a caustic agent. She was employed in a laundry and evidently she was desirous of avoiding work.

A male patient, aged seventy-two years, suffered from chronic pemphigus. Crops of bullæ were appearing daily on the trunk and the appendages; they had resisted all treatment and the patient was gradually becoming weaker.

A female, aged twenty-four years, had keloidal disfiguration of the face following an attack of erysipelas six months earlier. She stated that there had never been any break in the skin.

Genital Prolapse.

In the gynæcological unit of the hospital, DR. ROBERT FOWLER, DR. J. M. BUCHANAN, DR. H. G. FURNELL and DR. A. OLDHAM demonstrated a series of patients with varieties of genital prolapse, and a parallel series cured of similar conditions by various vaginal operations. Abdominal interventions were not advised for prolapse.

Lantern slides and a cinematographic exhibit further illustrated the features of the discussion.

Pulmonary Endarteritis.

DR. GLEN BUCKLE, on behalf of Dr. W. J. Penfold and himself, showed blood cultures from a female patient, aged eighteen years, who had been admitted to hospital complaining that she had felt off colour for two weeks. She had been in good health previously, except for growing pains in childhood. The only relevant signs which were elicited on examination were: flushed appearance of the face; rise in temperature to 39·1° C. (102·4° F.); pulse rate, 120 per minute (good volume); respirations, 24 per minute; blood pressure, systolic 140, diastolic 62 millimetres of mercury. The heart sounds were regular, no right cardiac dulness was apparent, the apex beat was palpable 10·0 centimetres (four inches) from the mid-line, a soft blowing systolic bruit and a systolic thrill were audible at the apex; a harsh systolic murmur at the pulmonary area was heard, also at the aortic area; a diastolic bruit was heard to the left of the sternum, and the pulmonary second sound was accentuated; a few rhonchi and crepitations were present at the base of each lung.

The patient died after having been in hospital for three weeks. During the first sixteen days her general condition was practically unchanged, the temperature swinging between 37·2° and 40·5° C. (99° and 105° F.) daily, and the systolic blood pressure steadily falling to 96 millimetres of mercury. A purpuric rash appeared on the abdomen on the eleventh day and on the side of the chest on the sixteenth day. During this period indifferent streptococci (not producing hæmolysis or a green colour on blood agar) were recovered from the blood on three successive occasions. After the sixteenth day the condition of the patient became rapidly lower; hæmolytic streptococci, *Streptococcus viridans* and indifferent streptococci were cultured from the blood.

At the *post mortem* examination the endocardium and the heart valves were found to be undamaged, but a vegetative lesion was present in the pulmonary artery, starting just above the valve and continuing upward to the region of the bifurcation of the artery. From the vegetation and from the spleen indifferent streptococci, hæmolytic streptococci and *Streptococcus viridans* were recovered. The cultural appearances of these types of streptococci were demonstrated.

Immunity to Cysticercus Bovis.

DR. W. J. and DR. H. B. PENFOLD and MISS MARY PHILLIPS, B.Sc., exhibited specimens and photographs to illustrate a solid active immunity in the ox to *Cysticercus bovis*. It was explained that *Cysticercus bovis* was the name given to the disease in oxen produced by the larval stage of *Tænia saginata*. The ox acquired the disease by ingesting the ova of the adult worm. Beef so affected was spoken of as "measly beef". In Victoria *Tænia saginata*, though rare, was the commonest of the tapeworms infecting man.

The active immunity experiment was the first of its kind to be performed. It showed that, with time, in artificially infested cattle the infestation entirely disappeared and, in addition, these cattle were immune to further infestation. The immunity to the disease apparently showed itself in two ways: (i) as it developed during the primary infestation it killed the cysticerci and so promoted their absorption; (ii) after it had developed as the result of the primary infestation it protected the oxen from further infestation, even when a large number of viable *Tænia saginata* ova were ingested.

Tissue Cultures of Human Brain Tumour Cells.

MISS M. CRANAGE and DR. J. SUTHERLAND showed, under the microscope on a warm stage, migrating astrocytes and other cells growing in tissue cultures of material taken two weeks previously, at operation, from an astrocytoma. The cultures were of the "hanging drop" type, the culture medium consisting of a mixture of chick embryo extract in tyrode solution with hen and human blood plasma. The cultures had been maintained by incubating at 37° C. and refeeding every second or third day with fresh culture medium. In this way single cells which had migrated out from the original implant of tumour tissue could be observed individually in the living active form.

Cultures were also shown which were vitally stained, as well as others which were permanently fixed and stained by the usual histological methods.

It was pointed out that this work was being carried out at the Baker Institute for and in collaboration with Dr. Leonard Cox and Dr. Hugh Trumble, in connexion with their brain tumour surgery at the Alfred Hospital. It was hoped that the tissue culture of tumour cells of the brain might be of use in identifying the type of tumour and in studying the behaviour of the living tumour cells *in vitro*.

(*To be continued.*)

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Lowe, Jack West, M.B., B.S., 1933 (Univ. Sydney), 2, Dennison Hall, Marcel Avenue, Randwick.

Obituary.

LEILA KATHERINE KEATINGE.

WE regret to announce the death of Dr. Leila Katherine Keatinge, which occurred on January 4, 1936, at Sydney, New South Wales.

Correspondence.

PROBLEMS OF JUNIOR RESIDENT MEDICAL OFFICERS.

SIR: The article written by Dr. Brodsky (THE MEDICAL JOURNAL OF AUSTRALIA, December 28, 1935) contains a great deal of truth and should receive attention at the appropriate quarter. The junior residents at the larger metropolitan hospitals seem to be nobody's interest and are such a disorganized amorphous group as to command no spokesman and no urge for redress. If they unite and form such an association as Dr. Brodsky suggests, they will doubtless come into their own—more remuneration, better recognition, better housing, better leave, and, most needed of all, better respect. The tendency at some hospitals seems to be to subjugate these young graduates, even as they were vassals during their six years of academic training.

My own view is that the British Medical Association should tackle this question of remuneration for and on behalf of these neither fowl-flesh-nor-good-red-herring young graduates. Surely there is some committee of the British Medical Association which would fight for fair conditions. No one wants to see these young men and maids go on strike, yet they will command little or no respect if they continue to sit down and take all that is coming to them in the progressive manner of the last few years. Some greybeard of the profession, some Macquarie Street Hampden, might well do battle for these youngsters, and it is with that conviction that I write these few lines.

It occurs to me that there is a great weakness in the education of the medical graduate. As an undergraduate he is mere clay in the hands of the potter and is moulded by the fingers and brains of countless professors, lecturers and teachers. As a graduate well out in the world he is catered for by the Post-Graduate Committee, which is now to be under the ægis of the University. But, as the recent graduate and while in residence at the hospitals, he is nobody's darling. It is just the time and place when he might be taught most, and it is the business of the Post-Graduate Committee or of the Faculty of Medicine (or the University) to undertake a scheme of supervision and direction of the training of these young graduates. They will never be given much practical surgery, if only because honoraries are too fearful of a yellow Press sensationalizing a death which might occur, and because there is a school of prevalent thought that it were injudicious to give these young residents much surgical experience lest they go out into the world and practise it! Dr. Brodsky is as one crying in the wilderness, with multiple and genuine wrongs and grievances to voice, and I trust I may help him towards their recognition and solution by these comments.

Yours, etc.,

"SENIOR."

January 2, 1936.

Books Received.

ELEMENTARY HYGIENE FOR NURSES: A HANDBOOK FOR NURSES AND OTHERS, by H. C. Rutherford Darling, M.D., M.S., F.R.C.S., F.R.F.P.S.; Sixth Edition; 1935. London: J. and A. Churchill. Crown 8vo, pp. 358, with illustrations.

THE PATHOLOGY OF INTERNAL DISEASES, by W. Boyd, M.D., M.R.C.P.E., F.R.C.P., Dipl.Psych., F.R.S.; Second Edition; 1935. Philadelphia: Lea and Febiger. Royal 8vo, pp. 904, with 335 illustrations. Price: $10.00 net.

CHRONIC STREPTOCOCCAL TOXÆMIA AND RHEUMATISM, by J. D. Hindley-Smith, M.A., M.R.C.S., L.R.C.P.; 1935. London: H. K. Lewis and Company, Limited. Crown 8vo, pp. 288. Price: 7s. 6d. net.

A CLINICAL TEXT-BOOK OF TROPICAL MEDICINE, by C. D. de Langen and A. Lichtenstein, translated from the Dutch by A. H. Hamilton, B.A., M.D., L.M.B.D.; 1936. Royal 8vo, pp. 557, with illustrations. Price: 27s. net.

PRACTICAL CLINICAL PSYCHIATRY FOR STUDENTS AND PRACTITIONERS, by E. A. Strecker, A.M., Sc.D., M.D., and F. G. Ebaugh, A.B., M.D.; Fourth Edition, rewritten and enlarged; 1935. Philadelphia: P. Blakiston's Son and Company. Demy 8vo, pp. 723, with 60 illustrations. Price: $5.00 net.

POCKET MONOGRAPHS ON PRACTICAL MEDICINE: THE EARLY DIAGNOSIS OF MALIGNANT DISEASE, by G. Keynes, M.A., M.D., F.R.C.S.; 1935. London: John Bale, Sons and Danielsson, Limited. Foolscap 8vo, pp. 70, with illustrations. Price: 2s. 6d. net.

THE MODERN TREATMENT OF BURNS AND SCALDS, by P. H. Mitchiner, M.D., M.S., F.R.C.S.; 1935. London: Baillière, Tindall and Cox. Demy 8vo, pp. 72, with 12 plates. Price: 5s. net.

INCOMPATIBILITY IN PRESCRIPTIONS AND HOW TO AVOID IT (WITH A DICTIONARY OF INCOMPATIBLES), by T. Stephenson, D.Sc., Ph.C., F.R.S.E., F.C.S.; Fourth Edition; 1935. Edinburgh: The Prescriber Offices. Demy 8vo, pp. 69.

THE DESIGN OF EXPERIMENTS, by R. A. Fisher, Sc.D., F.R.S.; 1935. Edinburgh: Oliver and Boyd. Demy 8vo, pp. 262. Price: 12s. 6d. net.

THE ESSENTIALS OF LIGHT TREATMENT FOR THE USE OF MASSEUSES, by E. M. Anderson, M.D., B.S., with foreword by P. Hall, M.R.C.S., L.R.C.P.; 1935. London: Baillière, Tindall and Cox. Demy 8vo, pp. 93. Price: 5s. net.

AIDS TO MEDICINE, by J. L. Livingstone; Fifth Edition; 1935. London: Baillière, Tindall and Cox. Foolscap 8vo, pp. 424. Price: 5s. net.

TONSILS AND NASO-PHARYNGEAL SEPSIS, by E. A. Peters, M.D., F.R.C.S.; 1935. London: Baillière, Tindall and Cox. Crown 8vo, pp. 99, with illustrations. Price: 5s. net.

RÖNTGENOLOGY: THE BORDERLANDS OF THE NORMAL AND EARLY PATHOLOGICAL IN THE SKIAGRAM, by A. Köhler; Second English Edition, translated by A. Turnbull, M.A., B.Sc., M.B., Ch.B.; 1935. London: Baillière, Tindall and Cox. Imperial 8vo, pp. 698, with 400 illustrations. Price: 50s. net.

ÜBER DIE RHYTHMIK DER LEBERFUNKTION, DES STOFFWECHSELS UND DES SCHLAFES, by E. Forsgren; 1935. Stockholm: Isaac Marcus Boktryckeri-Aktiebolag. Imperial 8vo, pp. 56, with illustrations.

AN INTRODUCTION TO SURGERY, by R. Morison, M.D., F.R.C.S., M.A., D.C.L., LL.D., and C. F. M. Saint, M.D., M.S., F.R.C.S., F.R.A.C.S.; Third Edition; 1935. Bristol: John Wright and Sons, Limited. Demy 8vo, pp. 377, with illustrations. Price: 15s. net.

A HANDBOOK OF BACTERIOLOGY FOR STUDENTS AND PRACTITIONERS OF MEDICINE, by J. W. Bigger, M.D., Sc.D., F.R.C.P.I., D.P.H., M.R.I.A.; Fourth Edition; 1935. London: Baillière, Tindall and Cox. Demy 8vo, pp. 474, with illustrations. Price: 12s. 6d. net.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY, by H. Bailey, F.R.C.S.; Fifth Edition; 1935. Bristol: John Wright and Sons, Limited. Royal 8vo, pp. 299, with illustrations. Price: 21s. net.

THE TREATMENT OF ACUTE POISONING, by H. L. Marriott, M.D., M.R.C.P.; 1935. London: John Murray. Demy 8vo, pp. 57, with illustrations. Price: 5s. net.

THE HAIR AND SCALP: A CLINICAL STUDY (WITH A CHAPTER ON HIRSUTIES), by A. Savill, M.A., M.D., M.R.C.P.I.; 1935. London: Edward Arnold and Company. Demy 8vo, pp. 295, with illustrations. Price: 12s. 6d. net.

ROYAT TREATMENT IN CARDIOVASCULAR DISEASE, by P. N. Deschamps, M.D.; 1935. London: Baillière, Tindall and Cox. Demy 8vo, pp. 119. Price: 5s. net.

MEDICAL AND D.P.H. EXAMINATION PAPERS, 1932-1935; 1935. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 233. Price: 3s. 6d. net.

RECENT ADVANCES IN LARYNGOLOGY AND OTOLOGY, by R. S. Stevenson, M.D., Ch.B., F.R.C.S.; 1935. London: J. and A. Churchill. Demy 8vo, pp. 356, with illustrations. Price: 15s. net.

FUNDAMENTALS OF BIOCHEMISTRY IN RELATION TO HUMAN PHYSIOLOGY, by T. R. Parsons, B.Sc., M.A.; Fifth Edition; 1935. Cambridge: W. Heffer and Sons, Limited. Crown 8vo, pp. 465. Price: 10s. 6d. net.

POST MORTEMS AND MORBID ANATOMY, by T. Shennan, M.D., F.R.C.S.; Third Edition; 1935. London: Edward Arnold and Company. Demy 8vo, pp. 724, with illustrations. Price: 30s. net.

Medical Appointments.

Dr. E. W. Turner has been appointed, pursuant to the provisions of the *Workers' Compensation Act*, 1928, to be a Certifying Medical Practitioner at Golden Square, Victoria.

* * *

Dr. H. E. Pellew has been appointed Temporary Honorary Clinical Assistant to the Gynæcological Section, Adelaide Hospital, South Australia.

* * *

Dr. B. S. Hanson has been appointed Honorary Clinical Assistant to the X Ray Department, Adelaide Hospital, South Australia.

* * *

Dr. C. A. Finlayson and Dr. J. S. Proctor have been appointed Official Visitors to the Mental Hospital at Parkside, South Australia, under the provisions of the *Mental Defectives Act*, 1913.

* * *

Dr. H. H. E. Russell has been appointed a Member of the Metropolitan Infectious Diseases Hospital Board, under the provisions of the *Metropolitan Infectious Diseases Hospital Act*, 1922, South Australia.

* * *

The undermentioned have been appointed to the Junior Resident Medical Staff of the Perth Hospital, Western Australia: Dr. J. B. L. Johnston, Dr. A. Beaumont, Dr. A. J. King, Dr. P. J. Orton, Dr. H. K. Pawsey, Dr. D. B. Skewes, Dr. H. N. Merrington, Dr. F. H. Lord, and Dr. C. E. Ryan.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xx.

ADELAIDE HOSPITAL, ADELAIDE, SOUTH AUSTRALIA: Registrars.

AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Honorary Officers.

DEPARTMENT OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Medical Officer.

LIDCOMBE STATE HOSPITAL AND HOME, LIDCOMBE, NEW SOUTH WALES: Honorary Dermatologist.

MENTAL HOSPITALS DEPARTMENT, PERTH, WESTERN AUSTRALIA: Assistant Medical Officer.

ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Senior Resident Medical Officer.

WESTERN AUSTRALIAN PUBLIC SERVICE: Medical Officer of Schools.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Richmond District Hospital, North Queensland. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY Hospital, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Welington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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